U.S. DEPARTMENT OF COMMERCE
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PB-251 438

Literature Study of Selected Potential Environmental Contaminants, Antimony and Its Compounds

Arthur D. Little, Inc.

Prepared For
Environmental Protection Agency

February 1976

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EPA-560/2-76-002

PB 251 438

LITERATURE STUDY OF SELECTED POTENTIAL ENVIRONMENTAL CONTAMINANTS ANTIMONY AND ITS COMPOUNDS

FEBRUARY 1976

FINAL REPORT

ENVIRONMENTAL PROTECTION AGENCY
OFFICE OF TOXIC SUBSTANCES
WASHINGTON, D.C.

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TECHNICAL REPORT DATA

(Please read Instructions on the reverse before completing)

1. REPORT NO.
EPA-560/2-76-002

2.

3. RECIPIENT'S ACCESSION NO.

4. TITLE AND SUBTITLE
Literature Study of Selected Potential Environmental Contaminants
Antimony and its compounds

5. REPORT DATE
February 1976

6. PERFORMING ORGANIZATION CODE

7. AUTHOR(S)

8. PERFORMING ORGANIZATION REPORT NO.
C-78341

9. PERFORMING ORGANIZATION NAME AND ADDRESS
Arthur D. Little, Inc.
Acorn Park
Cambridge, Massachusetts 02140

10. PROGRAM ELEMENT NO.

11. CONTRACT/GRANT NO.
68-01-3198

12. SPONSORING AGENCY NAME AND ADDRESS
Office of Toxic Substances
Environmental Protection Agency
Washington, D.C. 20460

13. TYPE OF REPORT AND PERIOD COVERED
Final

14. SPONSORING AGENCY CODE

15. SUPPLEMENTARY NOTES

16. ABSTRACT

A comprehensive review of the literature published since 1 January 1954 was conducted to prepare this preliminary investigation report on the physical and chemical properties of antimony (Sb) and its compounds, on the environmental factors related to their consumption and use, on the health and environmental effects resulting from exposure to Sb and antimonials, and on the regulations and standards governing their use.

Antimony and its compounds, although not ranked among the biggest volume chemicals in the U.S., are industrially significant because of their contribution to the manufacture of many commonly-used products, such as alloys, paint, paper, plastics and textiles. Antimony trioxide (antimony oxide) is used to impart flame resistance to plastics, textiles, and other products. Antimony dusts have been associated with the development of pneumoconiosis in miners and other workmen. Some antimony compounds are toxic, or give rise to toxic decomposition products, but no serious occupational poisoning has been associated with the industrial use of antimony.

17. KEY WORDS AND DOCUMENT ANALYSIS

a. DESCRIPTORS	b. IDENTIFIERS/OPEN ENDED TERMS	c. COSATI Field/Group
Antimony Antimony Compounds Antimony Trioxide Environmental Contaminants Flame Retardants Tartar Emetic		

PRICES SUBJECT TO CHANGE

18. DISTRIBUTION STATEMENT

19. SECURITY CLASS *(This Report)*

20. SECURITY CLASS *(This page)*

21. NO. OF PAGES
163

22. PRICE
$6.75-2.25

EPA Form 2220-1 (9-73)

EPA-560/2-76-002

LITERATURE STUDY
OF
SELECTED POTENTIAL
ENVIRONMENTAL CONTAMINANTS

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Contract No. 68-01-3198

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Environmental Protection Agency
Washington, D.C. 20460

Frank Letkiewicz
Project Officer

Arthur D. Little, Inc.
Acorn Park
Cambridge, Mass. 02140
C-78341

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SUMMARY

Antimony, a metallic element, was known and used in ancient times as a metal in artifacts and jewelry. An antimony compound, antimony sulfide, served the early Egyptians as a cosmetic and the ancient Romans used antimony preparations as drugs. Although certain antimonials are still used as medicinals, such use is relatively minor. The major use for antimony metal is in antimonial lead, while its principal compound, antimony oxide, finds increasing use in the preparation of flame-retardant chemicals for plastics, textiles and other products.

Antimony, found primarily in mineral ores, is derived primarily from stibnite (antimony trisulfide), a mineral rich in antimony. Other natural sources of antimony are complex sulfide ore, which are ores of stibnite containing chiefly lead, copper, silver, and mercury. China has the world's largest antimony deposits, with known reserves amounting to almost 4 million tons. U.S. resources are very small, approximating 100,000 tons in known reserves, but the U.S. may have another 100,000 tons in undiscovered resources. A new find of antimony ore in the U.S. was reported in 1970, and the ore is currently being processed.

I. PHYSICAL AND CHEMICAL PROPERTIES

Metallic antimony is a hard, silver-white lustrous metal, which is relatively inert and resistant to tarnish. Although it is too brittle to be used as a pure metal, antimony alloys with lead and other metals, imparting desirable hardness to them.

Numerous antimony compounds exist and they are used industrially in storage batteries; miscellaneous chemical products; rubber and plastics; industrial chemicals, including inorganic pigments; stone, clay, and glass; power transmission, etc.

Antimony combines with sulfur to form antimony trisulfide or antimony pentasulfide. It reacts with chlorine to form antimony chlorides. Under controlled conditions it reacts with oxygen to form the oxides.

Antimony trioxide, known in the trade as antimony oxide, is the most important oxide of antimony. A white or colorless powder, its ability to suppress the chalking tendency of other white pigments makes it useful as a pigment. Its flame-retardant properties have contributed to its greatest commercial demand. Because it is insoluble, antimony oxide is particularly useful as a flame retardant in the paper industry.

II. ENVIRONMENTAL EXPOSURE FACTORS

A. Production and Consumption

Although antimony and its compounds are industrially significant because of their unique contribution to the manufacture of many commonly-used products, such as alloys, paint, paper, plastics, and textiles, they are not ranked among the biggest volume chemicals in the U.S. Total production in 1974 of primary and secondary antimony was only about 41,000 tons, with the secondary production (i.e., antimony produced from scrap rather than from its ores or as a by-product of lead smelting operations) accounting for better than 50% of total production.

The United States mine production of antimony is a very small part of world mine production. In 1974, the U.S. contributed only 661 short tons to the total world mine production of 76,419 short tons. In recent years, U.S. imports of antimony ores (Sb content) have been about 17,000 tons.

There are no U.S. or world production figures available for antimony oxide, but virtually all antimony oxide producers depend upon antimony ores for their operations. The availability of these ores governs the production of antimony oxide, as well as that of metallic antimony.

The two major producers of antimony in the U.S. are NL Industries, which processes ores received from Mexico in its smelter at Laredo, Texas, producing antimony and antimony oxide, and the Sunshine Mining Company in Kellogg, Idaho, which produces antimony from its own silver-copper ores by a process identified as leaching and electrolysis. Utilizing a new entimony find, the United States Antimony Corporation converts antimony sulfide concentrates to metallic antimony in a smelter at Thompson Falls, Montana.

Asarco, an established producer of antimony oxide derived as a by-product in the smelting of lead ores, recently announced plans to build an antimony plant in El Paso, Texas, that will have a capacity of 1,825-ton/year. Other antimony oxide producers include Chemetron Corporation, Harshaw Chemical Company, McGean Chemical Company, and M & T Chemicals. In 1974, Associated Metals and Minerals started operations in a new plant in Texas City, Texas, believed to have a capacity of at least 1.8 million lb/year.

In the production of primary antimony (as metal, oxide, or sulfide) from its ores the choice of production method depends upon the amount of antimony present in the ores. High-grade ores contain 45 to 60% antimony and these ores are usually processed by liquation. U.S. ores are of a lower grade and are smelted in a blast furnace, or are treated by leaching (using sodium hydroxide) and then electrolyzed to produce a 93 to 99% pure antimony. Impure metal is also refined by an electrolysis process to produce a 99.9% pure antimony metal.

In the smelting of antimony ores (and lead ores) the antimony sulfide is converted to antimony oxide, which is volatile. This material, generally impure, is captured in baghouses or other recovery devices and is further smelted to produce a commercially acceptable oxide.

Antimony has been a cyclic marketplace performer, its price remaining quite stable over the years. Its price rose sharply in recent years because of tight supplies, but are now trending downward again. The price drop is attributed to the depressed economy, particularly in the automotive field (which cut down battery production) and to the use of stockpiled antimony. Federal legislation requiring certain textiles and other materials to be flame proofed is expected, however, to boost the demand for antimony. Consumption should rise at an annual rate of 4% through 1985.

Antimony is used in metal and non-metal products. As a metal, it finds a use in antimonial lead, solder type metals, ammunition (bullets) storage batteries, and in cable coverings. As a non-metal, it is used in glass and clay products in flameproofing chemicals and compounds, in ammunition (primers), in fireworks, and in plastics and rubbers.

Antimony oxide as well as antimony pentasulfide and antimony trisulfide, is used in various grades to impart flame resistance to many rubber, plastic, textile, paint and paper products. The oxide is used as a pigment, primarily in paints. As a decolorizer, antimony oxide is useful in glass, particularly optical glass. It is also used as an opacifying agent in porcelainized enamel. The trifluoride is used in the manufacture of pottery, while the tri- and pentasulfides are used to make fireworks and matches.

Antimony oxide and other antimony compounds are also used as catalysts in the manufacture of other chemicals, the oxide serving as a catalyst, for instance in the production of polyester resins.

The medicinal use of antimony compounds has decreased in recent years, primarily because of undesirable side effects of some of these compounds or their toxicity, but also, because of the advent of new type drugs. Antimony potassium tartrate is an effective emetic and has been used to treat certain tropical diseases, but other less toxic antimony compounds are replacing it. The barium, as well as the potassium tartrate, is used as a veterinary medicinal.

Antimony is a significantly useful commercial product but it is not an essential one. Although it has certain properties that promote its use (e.g., solidified from the molten state, it expands on cooling; thus it offers a unique property for type metal used in printing), antimony can be replaced by other materials. Alternative compounds have been used, when military needs required the major part of production or when less expensive materials have been required.

In general, antimony is not present in significant amounts in air but, when emitted from smeltering operations, it has been measured in concentrations higher than its permissible Threshold Value Limit. Antimony emitted from smelters can, evidently, be carried for some distance by the wind.

Antimony is present in sea water at a normal concentration of 4 μg/l, but no excessive concentration of antimony in sea water (as the result of extracting minerals from the seabed or from waste disposal) has been reported. Because most salts of antimony are insoluble, there is little chance that large concentrations of antimony will be found in the ocean. Marine animals, however, do concentrate antimony in their muscles, and several studies indicate that certain levels of antimony have been toxic to some fish. Because of the possible consequences to human health that could result from eating seafood contaminated with high concentrations of antimony, it is advisable that any future large-scale extraction of antimony from the seabed be closely monitored. As little as 97 mg of antimony have been reported as lethal to humans.

The mining and crushing antimony ores offers potential for the generattion of antimony dusts but, especially in recent years, carefully controlled operations and the use of dust collecting systems have minimized the dust hazards.

In the production of antimony metal from its ores, by-product dust and antimony oxide fume can be generated. Again, however, most large industries use local exhaust systems in the working areas and provide a direct exhaust for the smelting furnace, thus minimizing hazards to employees and controlling air emissions from the plant.

Air emissions at the antimony smelter in Laredo, Texas, were reported to be higher than permissible in 1972, but the company has installed additional baghouses, hoods and controls, which have helped to reduce these emissions considerably. This smelter produces only an insignificant amount of liquid effluent, but does generate about 44,000 tons of solid waste (slag) each year. This slag, which contains 1% of antimony, is stored outdoors on company property.

In Idaho, the leaching and electrolysis process used by one company to produce antimony generates no air emissions or solid waste. A recycling system installed a few years ago has reduced the antimony content of the liquid to a very low amount. Currently, although antimony content is only 1.0 to 1.3 ppm, it is still too high to meet the suggested EPA proposed effluent limitations. The company is striving now to comply with the EPA requirements. The antimony effluent has not had any deleterious effect on vegetation in the area, but fish have not been able to survive in the local river. This effect, however, has not been attributed to the presence of antimony in the effluent.

In general, there is no evidence that antimony oxide is a harmful pollutant. It is discarded into municipal systems.

Exposure to antimony dust and its salts can cause dermatitis. Antimony vapors, when inhaled, can cause respiratory and gastrointestinal problems. Antimony dust or vapor, under certain conditions, can also be a fire hazard. For these reasons, particular antimony compounds have to be transported according to prescribed procedures. Storage areas should be dry, well-ventilated, and protected from heat and sunlight. Fire extinguishers should be kept in the storage area.

The potential dangers from excessive dust and fumes that exist in the production of antimony also exist in those industries where antimony is used. For example, antimony dust may be generated during the machining and polishing of antimony-containing metal products. Antimony trioxide dust may form when the oxide is used as a pigment in the manufacture of paints and other products; antimony pentoxide dust may be generated in the making of glass and ceramics. Other hazards may exist from skin contact with antimony trichloride solutions during the dyeing of textiles or with antimony sulfide in rubber compounding operations.

Federal, state and local regulations govern the disposal of antimony wastes. Landfill and smelter are the principal disposal methods now used; ocean dumping has been practiced, but concern over the potential hazard to the ecosystem has practically ruled out this method. Some antimony compounds, such as antimony pentafluoride and antimony trifluoride, must be pretreated chemically before being buried. Encapsulation of nickel antimonide before disposing of it in a chemical waste landfill is recommended. Because the antimony halides, e.g., antimony trichloride can be decomposed by water to form toxic hydrogen chloride, care must be exercised in their disposal.

Traditional laboratory analytical methods are used for determining the concentration of antimony in ores, minerals and dusts. Trace analysis of antimony and its compounds in air, in water and in biological specimens requires the greater sensitivity of modern instruments, such as atomic absorption spectroscopy.

Although there is an absence of environmental monitoring in operation for heavy metals as marine pollutants, schemes for automated analysis have been suggested using existing equipment. Minute amounts of antimony in the air of a few urban and nonurban areas have been recorded by the National Air Sampling Network. Methods for determining airborne particulate antimony are under development at Arthur D. Little Inc., for NIOSH.

III. HEALTH AND ENVIRONMENTAL EFFECTS

Antimony is found only infrequently in air, but following emission from an antimony (or copper or lead) smelter the antimony may be transported for some distance. There is evidence that it is persistent in surface soils following contamination. There is also abundant evidence of antimony uptake in human and animal tissues and organs with exposure to antimony, but some attempts to correlate tissue with age have been unsuccessful. Thus, it is uncertain whether antimony is actually accumulated. There is currently no evidence of biomagnification.

Occupational exposures to antimony ore, pure antimony, and antimony oxide have been implicated in increased incidence in workmen of pneumoconiosis, a disease of the lungs caused by the habitual inhalation of irritant minerals or metallic particles. In animal studies acute and subacute exposure to these forms of antimony resulted in a pulmonary phagocytic response (cell engulfing of foreign material) generally without any appreciable pneumonitis. With chronic exposure the macrophage (large phagocyte) response was more involved and there was an increase in fibrous tissue. In contrast, occupational exposure to antimony trisulfide has been associated with cardiovascular changes. Experimental studies with this compound in rats, rabbits and dogs confirmed the cardiovascular effects. Exposure to gaseous forms of antimony produced more generalized systemic toxicity and, in the cases of antimony trichloride in men and antimony pentafluoride in rodents, the effects were probably attributable to both the antimony and the halide components.

Trivalent antimonials are very effective therapeutic agents for treatment of schistosomiasis. A major side effect of this use has been cardiovascular changes which have been demonstrated electrocardiographically following administration of antimony dimercaptosuccinate (TWSb), potassium antimony tartrate (tartar emetic), sodium antimony tartrate, sodium antimony bis (pyrocatechol-2,4-disulfonate) and sodium antimonyl gluconate. The ECG changes were more marked with TWSb than tartar emetic in man. No similar changes have been reported following administration of pentavalent antimonials. The mechanism of the cardiovascular effect has not been clearly defined. Trivalent antimonials have also been demonstrated to reduce contractile force in intact and isolated dog hearts with tartar emetic producing a more pronounced effect than TWSb. At lower than therapeutic doses effects in animals were minimal. There is currently no evidence of carcinogenic, mutagenic or teratogenic effects, attributed directly to antimony or its compounds. Liver toxicity does not appear to be prominent although the highest tissue retention was found in liver in studies in animals.

In conclusion, the comparatively small annual production of antimony and its compounds in the United States coupled with the relatively safe history of the use of these materials indicates that they are not a major environmental contamination hazard. No serious occupational poisoning has been associated with the industrial use of antimony. Although some antimony compounds are toxic, or give rise to toxic decomposition products, many of them are used in small enough quantities to preclude the possibility of any large-scale hazard. Industrial safety measures imposed on industry by government regulations or adopted by manufacturers for their own corporate protection, or the health and safety of their employees, suggest that certain potential hazards are being properly addressed. Antimony in the general air environment does not appear to be a contaminant of great magnitude. Major U.S. rivers do not have excessive concentrations of antimony, and ocean dumping restrictions will curtail or eliminate the potential hazard to marine life, thus protecting man from the possible ill effects of eating fish contaminated with a concentration of antimony high enough to be hazardous or lethal. Nevertheless, wherever large-scale operations involving antimony or its products are conducted, careful monitoring of gaseous emissions and water pollutants should be encouraged.

PRELIMINARY INVESTIGATION REPORT

0. INTRODUCTION

A. History

Antimony, the metallic element identified by the chemical symbol Sb, an important industrial commodity in today's highly technical world, has been known to man for 6000 or more years. The literature refers to the early use of antimony and its sulfide as a cosmetic, a medicinal, and as a metal in artifacts and jewelry. In early biblical times, for instance, it was used as an eyebrow paint, and references to its use as a cosmetic appear in early Chinese and Arabic writings. Egyptian copper articles thinly coated with antimony have been traced back to around 2500 B.C. A cast antimony vase unearthed at Tello, Chaldea, is believed to have been made in 4000 B.C. In another instance, bracelets and necklaces found in graves, dating back to the aeneolithic age, contained almost 100% Sb. Another early use was as a pigment, when antimony compounds were combined with lead to produce a yellow glass.

The ancient Romans used antimony preparations as drugs, and Stone Age Egyptians used it to treat eye diseases. By the time of Pliny (~50 AD), antimony was recognized as a medicinal and by the fifteenth century it was widely used as such. Because of its observed toxic effects, however, the Faculty of Physicians of Paris banned its use in the mid-sixteenth century. Then, in 1657, this ban was lifted, when, all else having failed, an antimony medicinal was given to the ailing King Louis XIV, who recovered from his illness. Antimony continued to be used medicinally, then, until the nineteenth century, when it again fell into disfavor (Harvey, 1960).

A century later, antimony compounds found another use -- as parasiticides, their use to control leishmaniasis representing a breakthrough in chemotherapy. Organic antimony compounds are still used as drugs today, especially to combat worm infestations, e.g., schistosomiasis and filariasis (Harvey, 1960).

There is no record of who first added antimony to lead to produce a strong, hard, anti-corrosive alloy, or when this occurred. In the mid-1500's, however, Sb was used as an alloying agent in metal bells. At that time, in addition to its use as an alloy, antimony was used as an ingredient for making pewter, glass, and metal mirrors; as a pigment; and as an ulcer drug.

The advent of the printing press may mark the beginning of antimony's extensive use as an industrial material. Early cast type was made of lead-antimony alloys, and antimony is still used in varying percentages in current type metals. Antimony later achieved a reputation as a strategic commodity, following the discovery in 1800 that it could be used to harden the lead used for shrapnel bullets, producing a brittle bullet that fragmented when the shell burst. This function contributed to a tremendous increase in the production of antimony during the period of World War I.

Another important industrial use for antimony dates from 1839, when Isaac Babbitt used an alloy containing antimony for bearing metals. The development of the lead storage battery in 1850 gave an added impetus to the use of antimony. About a century later, antimony was used extensively as a hardening agent in the lead sheath used on telephone and other cables. More recently, antimony (particularly its oxides) has found an increasing use as a fire-retarding agent.

As the demand for antimony increased, there have been developments in the extraction of antimony from its ores. Crude antimony can be produced from a rich ore (stibnite) by a liquation process or from the roasting of sulfide ores and the liquation of antimony ores was evidently practiced in the sixteenth century. The roast reduction process for producing the metal was introduced in the 1700's. By 1830, the reverberatory furnace was being used. Shortly before 1900, electrolytic antimony was first produced, but the process only became commercially important after 1940. The blast furnace smelting process was investigated about the time of World War I, and since 1930 this method has been widely used to extract metallic antimony from its concentrates.

B. Sources, Occurrence, and Resources

From 0.2 to 0.5 parts per million (ppm) of antimony exist in the earth's crust and from 0.1 to 1 ppm are found in igneous rock, while deep-sea clays contain 1 ppm. Minute amounts of antimony are found in sedimentary rocks, and the detection of small amounts of the element in marine animals indicates its presence in sea water.

Native metallic antimony is seldom found. The element exists primarily in mineral ores, over 100 of which are found in nature. Of these, the most important is the antimony mineral stibnite, or antimony trisulfide (Sb_2S_3), which is rich in antimony. Stibnite, when exposed to oxidation, is converted to the oxide. Generally less important commercially than the trisulfide, the oxides include stibiconite (the hydroxide), cervantite, valentite, senarmontite, and kermasite (the oxysulfide ore). Of these, however, kermasite and stibiconite have been worked in some world areas.

Other sources containing relatively large percentages of antimony are the complex sulfide ores, which are ores of stibnite containing lead, copper, silver, and mercury. Though these ores are lean in antimony, the metal is usually recovered as a by-product element when the ores are smelted for lead or copper. For example, tetrahedrite, the sulfide of copper and antimony contains up to 29% Sb; jamesonite, the sulfide of lead and antimony, contains 32-35%; and bournonite, the sulfide of copper, lead, and antimony, contains 25%. For the most economic production of high purity metal (99.6^+%), ores containing minimum amounts of impurities (Pb, As, Cu, Zn) are required.

The world's largest antimony resources are in China. Known deposits there total 3.8 million short tons, but there are estimates of additional resources that could increase China's total to 5.3 million tons. Bolivia's known resources are estimated at 420,000 tons. The Republic of South Africa and the USSR each have resources estimated at 300,000 tons. Other identified resources include Mexico (200,000 tons), Australia (150,000 tons), Turkey (120,000 tons), and Thailand (110,000 tons). Yugoslavia and the United States each have 100,000 tons, but the U.S. may have an additional estimated 100,000 tons in undiscovered resources (Miller, 1973). About 50% of total U.S. reserves are low-grade ores because they are found in complex lead-silver-copper and gold ores. In 1955, the only economically workable U.S. deposits were in Idaho, and in the last 40 or so years about 80% of the U.S. mine production of antimony has been from Idaho, where resources in the Coeur d'Alene district are believed to be adequate for continuous operation for the long future. Other U.S. deposits that have been worked are in the districts of Fairbanks and Kantishna in Alaska, where resources are estimated at about 10,000 tons. Alaska's antimony resources are low-grade types. Included among the estimated U.S. resources are about 50,000 tons of antimony present in lead ores in southeastern Missouri, the Tri-State District, and the Upper Mississippi Valley (Miller, 1973). In 1970, Agau Mines, Inc. reported that it had located a major antimony find with an estimated 330,000 tons of antimony ore available in two veins. At that time, Agau expected to start processing 100 tons/day of antimony ore in mid-August 1970 and anticipated an eventual processing capability of 1,800 tons/days.

I. PHYSICAL AND CHEMICAL DATA

A. Structure and Properties

1. Metallic Antimony

Antimony, Sb, is a hard silver-white lustrous, odorless metal with a scale-like structure. In powder form it is a lustrous, dark gray. It is ordinarily quite stable and very resistant to tarnish since it does not oxidize in dry air at room temperatures. Under controlled conditions it will react with oxygen to form the oxides. It combines with sulfur in all proportions to form the trisulfide or pentasulfide. Fluorides or fluocomplexes are formed by the action of hydrofluoric acid on many insoluble antimony compounds. It reacts with chlorine to form the antimony chlorides. It is insoluble in water and not affected by cold, dilute acids. Antimony has very poor electrical and thermal conductance.

When antimony is in the molten state it will attack other metals. Antimony is brittle and cannot be rolled, forged or drawn but must be cast. When solidified from the molten state the metal will expand on cooling, a unique property, which makes it very desirable for type metal used in printing. While its brittle properties limit its uses as a pure metal, alloyed with other metals it imparts such properties as improved hardness and lower melting point. Sb alloys particularly with the metals lead (Pb), bismuth (Bi), tin (Sn), copper (Cu), nickel (Ni), iron (Fe), and cobalt (Co) and forms chemical compounds with the last four metals.

Table I shows the pertinent properties of antimony along with the names and properties of its commercially important compounds. These data were compiled from a review of such general references as the Merck Index, 8th edition, 1968; Kirk-Othmer, "Encyclopedia of Chemical Technology," 2nd edition, Vol. 2, pp. 562-8, 1963; Gmelin's Handbuch der Anorganischen Chemie; "Chemical Week," Buyers Guide Issue, 1976; American Conference of Governmental Industrial Hygienists, "Threshold Limit Values--1971"; and various manufacturers technical bulletins and catalogs.

TABLE I

PROPERTIES OF ANTIMONY AND SELECTED ANTIMONY COMPOUNDS

COMPOUND	SYNONYMS	FORMULA	MOLECULAR WEIGHT	APPEARANCE AND ODOR	BOILING POINT °C 1 ATM	MELTING POINT, °C	SPECIFIC GRAVITY	SOLUBILITY in WATER, %	LD_{50}	DEGRADATION PRODUCTS	PURITY %	PRINCIPAL IMPURITIES
Antimony, metallic	Antimony regulus	Sb	121.75	White to grey solid No odor	1635 ± 8° *	630	6.6	Insoluble		Sb_2O_3	99.8	As, Cu, Fe, Pb
Antimony lactate		$Sb(C_3H_5O_3)_3$	388.8	White solid						Sb_2O_3, CO_2, H_2O		
Antimony oxychloride	Basic antimony chloride Powder of Algaroth Mercurius vitae	SbOCl	173.2	White solid No odor				Soluble		Sb_2O_3, HCl(or salts)		
Antimony pentachloride	Antimonic chloride Antimony perchloride Antimony(V) chloride	$SbCl_5$	299.1	Pale brown liquid Acrid odor	140	3.5	2.35	Reacts		Sb_2O_5, HCl(or salts)	99.0	As, Fe, Pb, SO_4
Antimony pentafluoride		SbF_5	216.7	Viscous liquid Acrid odor	143	7.0	3.15	Reacts		Sb_2O_5, HF(or salts)	99.0	
Antimony pentasulfide	Antimonic sulfide Antimony(V) sulfide Golden antimony sulfide Antimony persulfide Antimony red Antimonial saffron	Sb_2S_5	403.8	Yellow-orange solid No odor			3.78	Insoluble Reacts slowly	1.5g/kg, ip, rat	Sb_2O_5, H_2S(or salts)		
Antimony pentoxide	Antimonic oxide Antimony(V) oxide Stibic anhydride Antimonic acid	Sb_2O_5	323.5	Yellowish solid No odor			3.78	0.087	4g/kg ip, rat	Metal antimonates		
Antimony potassium tartrate	Tartar emetic Tartrated antimony Potassium antimonyl tartrate	$(SbO)KC_4H_4O_6$	324.9	White solid No odor			2.6	8.3	50mg/kg ip, mice; 600mg/kg, po, mice	Sb_2O_3, K salts, CO_2, H_2O	99.9	As, Pb

*Gmelin's Handbuch der Anorganischen Chemie gives a boiling point range of 1322-1635 ± 8°C for antimony.

TABLE I
PROPERTIES OF ANTIMONY AND SELECTED ANTIMONY COMPOUNDS (Cont'd)

COMPOUND	SYNONYMS	FORMULA	MOLE-CULAR WEIGHT	APPEARANCE AND ODOR	BOILING POINT °C 1 ATM	MELTING POINT, °C	SPECIFIC GRAVITY	SOLUBILITY in WATER, %	LD_{50}	DEGRADATION PRODUCTS	PURITY, %	PRINCIPAL IMPURITIES
Antimony sodium tartrate	Antimony, tartarized Sodium antimonyl tartrate Stibunal Emeto-Na	$(SbO)NaC_4H_4O_6$	308.8	White solid No odor				62	25mg/kg, iv, mice	Sb_2O_3,Na salts,CO_2,H_2O	98-101	As, Pb
Antimony sulfate	Antimous sulfate Antimony trisulfate Antimony(III) sulfate	$Sb_2(SO_4)_3$	531.7	White solid			3.62	Soluble	1g/kg, ip, rat	Sb_2O_3,sulfate salts	99	
Antimony tetroxide		Sb_2O_4	307.5	White to yellow solid			5.8	0.002			99.9	
Antimony tribromide		$SbBr_3$	361.5	White solid No odor	288	96	4.15	Reacts		Sb_2O_3,HBr(or salts)	99.9	
Antimony tri-n-butylate	Antimony tributoxide Tri-n-butyl antimonite	$(C_4H_9O)_3Sb$	341	Colorless liquid			1.28	Insoluble		Sb_2O_3,$C_4H_9OH(CO_2,H_2O)$	99	
Antimony trichloride	Butter of antimony Antimony(III) chloride Antimonous chloride Antimony chloride	$SbCl_3$	228.1	White solid Acrid odor	223	73	3.14	Reacts; very soluble		Sb_2O_3,HCl(or salts)	99.5	As, Fe, Cu
Antimony triethoxide	Antimony triethylate	$(C_2H_5O)_3Sb$	256	Colorless liquid	95 (at 11mm)		1.52	Insoluble		Sb_2O_3,$C_2H_5OH(CO_2,H_2O)$		
Antimony trifluoride	Antimonous fluoride Antimony fluoride Antimony(III) fluoride	SbF_3	178.8	White solid	376	292	4.38	443		Sb_2O_3,HF(or salts)	98	
Antimony triiodide	Antimony iodide Antimony(III) iodide	SbI_3	502.5	Red solid	401	168	4.92	Reacts		Sb_2O_3,HI(or salts)		

TABLE I

PROPERTIES OF ANTIMONY AND SELECTED ANTIMONY COMPOUNDS (Cont'd)

COMPOUND	SYNONYMS	FORMULA	MOLE-CULAR WEIGHT	APPEARANCE AND ODOR	BOILING POINT C 1 ATM	MELTING POINT C	SPECIFIC GRAVITY	SOLUBILITY in WATER, %	LD_{50}	DEGRADATION PRODUCTS	PURITY %	PRINCIPAL IMPURITIES
Antimony trioxide	Antimony oxide Antimony(III) oxide Diantimony trioxide Antimony bloom Flowers of antimony	Sb_2O_3	291.5	White solid No odor	1550	656	5.2	0.001	>20g/kg po, rat	Metal antimonites	99	As, Fe, Pb
Antimony trisulfide	Stibnite Antimony sulfide, native Antimony glance Needle anti antimony Antimony gray Antimony sesqui-sulfide	Sb_2S_3	339.7	Gray, black or red solid No odor	1150	550	4.64	0.0017	1g/kg, ip, rat			
Antimony-zinc(oxide mixture)		Sb_2O_3(12.5%) ZnO(12.5%		White solid No odor			3.3	Insoluble				
Sodium antimonate	Sodium meta-antimonate Leukonin	$NaSbO_3$	192.7	White solid				Insoluble		Insoluble antimony salts		
Stibine	Antimony hydride	SbH_3	124.8	Colorless gas Characteristic odor	-17	-88.5	2.2 (liquid)	0.5		Metallic antimony		

The following table includes additional properties of antimony.

PHYSICAL PROPERTIES OF HIGH PURITY ANTIMONY (99.9999%)

Atomic Number	51
Molecular Weight	121.75
Melting Point	630.5°C
Boiling Point	1635° ± 8°C
Density (20°C)	6.618 g/cc
Latent Heat of Fusion	38.3 cal/g
Latent Heat of Vaporization	16.1 cal/g
Specific Heat	0.0494 cal/g/°C
Thermal Conductivity	0.045 cal/sq. cm./cm./°C/sec
Resistivity (0°C)	39.1 microhm-cm
Standard Electrode Potential	+0.1 v
Magnetic Susceptibility	0.87 cgs
Crystal Structure	Rhombohedral
Thermal Neutron Cross Section	5.7 barns
Hardness, Moh's Scale	3.0

The principal contaminants of antimony are arsenic, copper, iron, and lead. The amount of impurities present in high-purity antimony are indicated below:

TYPICAL ANALYSIS

	99.9999%	99.999+%
As	<0.5 ppm	2 ppm
Fe	0.1 ppm	1 ppm
Pb	--	<1 ppm
Cu	--	1 ppm
Bi	--	<1 ppm
Others	<0.3 ppm	<2 ppm

The commercial product is generally sold in the following standard forms: semi-circular ingot, broken pieces, granules, and cast cake. Other forms are: powder, ingot, shot, and single crystals.

2. Antimony Compounds

The formulas and properties of the antimony compounds considered of commercial importance are listed in Table I.

Undoubtedly the most industrially important of these compounds is antimony trioxide, commonly called antimony oxide. A white or colorless powder, antimony trioxide continues to be in demand as a flame-retarding agent for many materials. Odorless, Sb_2O_3 is only very slightly soluble in water. It is soluble in aqueous hydrochloric acid, concentrated alkalies, potassium hydroxide, and acetic acid.

Antimony trichloride, a white solid with an acrid odor, also exists as colorless crystals or flakes that are hygroscopic. $SbCl_3$ fumes in air. It is very soluble in water and in absolute ethanol, hydrochloric acid, acetone, ether, benzene, chloroform, and tartaric acid. Hydrolysis yields antimonyl chloride (SbOCl). A number of ethers, aldehydes, and mercaptans will form one-to-one addition compounds with antimony chloride to produce organic compounds of antimony.

Antimony pentachloride is manufactured as a colorless to yellow liquid, which is soluble in hydrochloric acid, chloroform, and carbon tetrachloride. It is corrosive and fumes in air. Hydrates are formed in the presence of small amounts of water. Chlorine is readily lost from $SbCl_5$, thus making it useful as a chlorine carrier. Ethers, alcohols, aldehydes, esters, and nitriles form one-to-one adducts with this compound. It is a poor conductor of electricity. However, solutions of it in liquid SO_2 are conductors.

Antimony potassium tartrate occurs as transparent, odorless crystals (which effloresce on exposure to air) or a white crystalline powder. It has a sweetish metallic taste and is poisonous. It is slightly soluble in cold water, and readily soluble in hot water, but insoluble in alcohol.

Antimony pentoxide is produced in the form of a yellowish powder which is slightly soluble in water but soluble in strong bases forming antimonates. It dissolves slowly in warm hydrochloric acid or in warm potassium hydroxide solution. It reacts with hydrogen sulfide to form antimony(V) sulfide.

Antimony trisulfide occurs in nature as black crystalline stibnite but, when manufactured, it is produced as a gray lustrous crystalline mass or grayish black powder. There is also a red modification. It is insoluble in water but soluble in concentrated hydrochloric acid with the evolution of hydrogen sulfide. It is soluble in solutions of alkali hydroxides containing excess caustic.

Antimony pentasulfide, Sb_2S_5, is an orange-yellow odorless powder which is insoluble in water but soluble in concentrated HCl with the evolution of hydrogen sulfide. It is also soluble in solutions of alkali hydroxides or sulfides, forming sulfantimonates. Known also as golden antimony sulfide, antimonic sulfide, antimonial saffron, antimony red, and antimony persulfide, its density is 3.78; its molecular weight is 403.82; and it has an LD_{50} of 1.5 g/kg, ip in rats.

Kirk-Othmer (1963) lists, in addition to antimony potassium tartrate, a number of antimonials that have been used as medicinals. These include the antimony (III) compounds: antimony sodium tartrate, stibophen, antimony sodium thioglycollate, antimony thioglycollamide, antimony lithium thiomalate, and the antimony (V) compounds: stibamine glucoside, ethylstibamine, ureastibamine, stibenyl, stibosan, and antimony sodium gluconate.

Antimony sodium tartrate, $(SbO)NaC_4H_4O_6$, whose synonyms and properties are given in Table I, appears as transparent or whitish scales or powder, is hygroscopic, and is much more soluble than antimony potassium tartrate.

Stibophen, $C_{12}H_4Na_5O_{16}S_4Sb.7H_2O$, or sodium antimony(III)bis(pyrocatechol-2,4-sodium disulfonate), is also known as Fuadin, Sodium Antimosan, Neoantimosan, and Pyrostib. A colorless crystalline powder, it is soluble in water, and insoluble in alcohol and other organic solvents. The aqueous solution turns yellow and oxidizes in air. Stibophen has a molecular weight of 895.27.

Antimony sodium thioglycollate, $NaSb(SCH_2COO)_2$, or antimony sodium thioacetate, has a molecular weight of 324.96. Its hygroscopic crystals turn pink in light.

Antimony thioglycollamide, $Sb(SCH_2CONH_2)_3$, with a molecular weight of 392.09, melts at 140°C.

Antimony lithium thiomalate is also known as anthiomaline.

Stibamine glucoside, also called Neostam stibamine glucoside, is an impure or incompletely characterized compound.

Ethylstibamine, or Neostibosan, is a mixture of ρ-aminobenzenestibonic acid, ρ-acetamidobenzenestibonic acid, antimonic(V) acid, and diethylamine.

Ureastibamine, also known as stiburea, is sometimes identified as ρ-ureidobenzene-stibonic acid, but in reality it is a complex mixture whose composition varies.

Stibenyl, $CH_3CONHC_6H_4SbO_3HNa$, is sodium 3-chloro-4-acetamidobenzenestibonate.

The antimony sodium gluconate, $C_6H_8NaO_7Sb$, which is used medicinally, is the pentavalent antimony compound also known as Solustibosan, Myostibin, and Pentostam. It occurs as crystals and is freely soluble in water. A trivalent antimony sodium gluconate, known as Triostam, also exists. An amorphous powder, it is soluble in water; its molecular weight is 336.88.

Numerous other antimony compounds are mentioned in the literature, some particularly in discussions of their environmental health effects. (See: Section III). These include:

Antimony acetate, $Sb(OOCCH_3)_3$, a crystalline material with a molecular weight of 298.88 that decomposes on boiling.

Antimony acetylacetonate, $Sb(C_5H_7O_2)_3$, more properly called antimony acetonylacetate, whose molecular weight is 418.93.

Antimony aluminide, SbAl, an antimony alloy with a molecular weight of 418.93.

Antimony arsenide, Sb_2As_3, (SbAs); molecular weight is 468.25.

Antimony barium tartrate, $Ba[SbOC_4H_4O_6]_2$, described as a white, fluffy powder or precipitate.

Antimony dichlorotrifluoride, $SbCl_2F_3$, a viscous liquid with a molecular weight of 249.67.

Antimony iodosulfide, SbIS, which has a molecular weight of 280.72.

Antimony orthophosphate, $SbPO_4$, which has a molecular weight of 216.74.

Antimony pentaselenide, Sb_2Se_5, has a molecular weight of 638.30.

Antimony phosphide, SbP, has a molecular weight of 152.72.

Antimony potassium oxalate, $K_3[Sb(OOCCOO)_3]$, also identified as potassium trioxalatoantimonate(III) or potassium oxalatoantimonate(III), is sometimes referred to as "antimony salt." A crystalline powder, soluble in water, it has a molecular weight of 503.12.

Antimony silver selenide, $AgSbSe_2$, has a molecular weight of 387.56.

Antimony silver telluride, $AgSbTe_2$, has a molecular weight of 484.86.

Antimony tartrate, $Sb_2(C_4H_4O_6)_3 \cdot 6H_2O$, has a molecular weight of 795.8.

Antimony triselenide, Sb_2Se_3, is a gray powder with a melting point ranging from 572-617°C and a molecular weight of 480.40. It is very slightly soluble in water.

Antimony tritelluride, Sb_2Te_3, has a molecular weight of 626.35.

Astiban, See: Stibocaptate.

Stibocaptate, $C_{12}H_9K_3O_{12}S_6Sb_2$; 2,3-Dimercaptosuccinic acid cyclic thioantimonate (III) S,S-diester with 2,3-dimercaptosuccinic acid tripotassium salt; known also as "antimony dimercaptosuccinate", Astiban, TWSb, is a crystalline powder with a molecular weight of 898.42. (The *Merck Index* notes that Astiban was described as sodium antimony-2,3-meso-dimercaptosuccinate by Browne and Schulert in *Am. J. Tropic Med. Hyg.* vol. 13, 558 (1964)).

Some compounds are apparently in use or at least are available for sale, but there is no data on them. No data were found for such compounds as antimony fluoroborate; antimony(III) methoxide; antimony tritungstate, $Sb(WO_4)_3$; antimony oxalate; and piperazine diantimony tartrate. There were also no data for tributyl antimony, $(C_4H_9)_3Sb$, or triphenyl antimony, $(C_6H_5)_3Sb$, but the *Chemical and Process Technology Encyclopedia* (1974) lists triethyl antimony and trimethyl antimony with the following information. *Triethyl antimony*, $Sb(C_2H_5)_3$, melts at -29°C, boils at 159°C, and has a specific gravity of 1.3. Its formula weight is given as 208.88. It is insoluble in water, but soluble in alcohol and ether. *Trimethyl antimony*, $Sb(CH_3)_3$, has a formula weight of 166.83, a specific gravity of 1.5, and boils at 81°C. It is slightly soluble in water.

II. ENVIRONMENTAL EXPOSURE FACTORS

A. Production, Consumption

1. Production

a. U.S. Production

In 1974, the United States produced 40,888 short tons (metal content) of antimony, the second highest production in the last 16 years. From a low of 29,469 short tons in 1959, production rose gradually to a peak of 39,724 short tons in 1966. Production then declined slightly from 1967 to a low of 33,316 short tons in 1971, rising to 36,261 short tons in 1972 and peaking again to a high of 41,813 short tons in 1973.

Antimony production falls into two categories, primary and secondary. Sources of primary antimony include mine and smelter production. In 1974, 661 short tons (antimony content) were mined while primary smelter production of antimony was 16,657 short tons. Secondary production accounted for 23,570 short tons, virtually all of which was used with lead in batteries and hard lead alloys. In 1975, domestic mine output of antimony totalled 950 short tons, or 44% above 1974, but primary smelter production of antimony materials was about 13% below the 1974 level due to a shortage in feed material from both reduced imports and lower secondary smelter production from scrap.

Primary production includes antimony in the forms of the metal, oxide, sulfide, and as a by-product in the manufacture of lead, plus the residual unrecovered antimony in the processing of ores from antimonial minerals. Secondary production accounts for over half of the industrial usage in the United States and comes from scrap, drosses, and residues. Secondary recovery in 1973 was 85% from old scrap and 15% from new scrap (Wyche, 1973) with the following breakdown reported:

Sources of Secondary Antimony:

Old Scrap		85%
Batteries	66%	
Type Metal	14%	
Babbitt	12%	
Others	8%	
	100%	
New Scrap		15%
Drosses		
Residues		
		100%

Table II shows the U.S. production of primary and secondary antimony from 1959 through 1974 and Table III shows the first three quarters of 1975 compared to 1974.

U.S. antimony mine production capacity is expected to increase toward 1980 with smelter production continuing at the same level (Wyche, 1975a)

b. Imports

The United States is one of the smallest producers of antimony in the world with mine production 661 short tons (antimony content) in 1974 as compared to a total world mine production of 76,419 short tons. Imports into the U.S. make up the difference between domestic production and consumption.

In the world market the major producers are the Peoples Republic of China, the USSR, Bolivia and South Africa. The important small producers are Canada, Mexico, Yugoslavia, and Thailand. The U.S. imports antimony ores from several of these countries, with the leading suppliers including the Republic of South Africa, Bolivia, Chile, and Mexico. Table IV shows the principal sources and volumes of recent imports of antimony ore and concentrate.

TABLE II

U.S. PRODUCTION OF PRIMARY AND SECONDARY ANTIMONY

(As reported by U.S. Bureau of Mines, in short tons, antimony content)

	PRIMARY								
	Mine	Metal	Oxide	Sulfide	Residues	Byproduct Ant'l Lead	Total Primary	Secondary Antimony Produced from New and Old Scrap	Total
1959	678	2,667	4,411	70	430	1,170	9,426	20,043	29,469
1960	635	3,665	5,188	60	385	656	10,589	20,104	30,693
1961	689	4,558	4,609	84	355	1,723	12,018	19,466	31,484
1962	631	4,407	4,788	53	366	2,113	12,358	19,362	31,720
1963	645	4,160	5,983	76	392	1,506	12,762	20,803	33,565
1964	632	4,418	6,748	53	447	1,692	13,990	22,339	36,329
1965	845	4,216	6,485	94	205	1,389	13,234	24,321	37,555
1966	927	4,567	7,791	126	219	1,833	15,466	24,258	39,724
1967	892	4,002	6,612	71	249	1,532	13,358	23,664	37,002
1968	856	3,617	6,518	133	417	1,801	14,345	23,699	37,044
1969	938	3,129	7,746	95	330	1,903	14,141	23,840	37,981
1970	1130	3,732	8,261	23	384	981	14,511	21,424	35,935
1971	1025	3,816	6,272	18	136	1,132	12,399	20,917	33,316
1972	489	3,837	8,343	232	201	731	13,833	22,428	36,261
1973	545	2,859	11,273	92	1,839	1,143	17,751	24,062	41,813
1974[1]	661	3,030	10,445	54	2,066	1,062	17,318	23,570	40,888
1975[2]									

Source: Compiled from data in U.S. Bureau of Mines Minerals Yearbook, 1962-1973

1. 1974 figures compiled from data in U.S. Bureau of Mines, Mineral Industry Surveys, Dec. 18, 1975.
2. Table III contains figures for the first three quarters of 1975.

TABLE III

U.S. PRODUCTION

of

PRIMARY AND SECONDARY ANTIMONY

(Short Tons)

			1974	First Quarter 1975	Second Quarter 1975	Third Quarter 1975
PRIMARY						
	Mine		661	248	190	241
	SMELTER	Metal	3,030	857	746	442
		Oxide	10,445	1,712	1,550	1,539
		Sulfide	54	---	---	---
		Residues	2,066	257	150	145
		Antimonial lead	1,062	65	157	280
SECONDARY			23,570	3,572	2,948	3,392
TOTAL			40,888	6,711	5,741	6,039

Source: Division of Nonferrous Metals, U.S. Bureau of Mines, Mineral Industry Surveys. December 18, 1975.

TABLE IV

U.S. IMPORTS ANTIMONY ORE

General imports of antimony ore (metal content) by countries, as reported by the U.S. Bureau of Mines, in short tons.

	1970	1971	1972	1973	1974
Bolivia	2,162	1,593	2,562	3,662	2,669
Chile	42	311	1,722	1,590	2,283
Mexico	3,666	2,314	2,217	2,088	1,629
Rep. of South Africa	6,239	3,826	10,160	6,446	4,739
Turkey	.....	.....	.....	1,339	201
Other Countries ...	1,711	1,575	551	1,554	3,134
Total............	13,820	9,619	17,212	16,679	14,655

SOURCE: Compiled from U.S. Bureau of Mines Mineral Yearbook 1971-1973 and Division of Nonferrous Metals, Bureau of Mines, Mineral Industry Surveys, Third Quarter 1975.

As shown in Table IV, imports of antimony ore and concentrate amounted to 14,655 short tons antimony content in 1974. Other imports in 1974 from a number of other countries included 5,203 short tons (antimony content) of antimony oxide; 2,203 short tons of antimony metal; and 58 short tons of antimony as needle antimony or liquated antimony sulfide (crude antimony). (Wyche, 1975b)

2. Producers/Distributors

The only U.S. mine that currently operates primarily for antimony is owned by United States Antimony Corporation, a subsidiary of Agau Mines, Inc., and is located southwest of Thompson Falls, Montana. In 1973, the only other source of domestic antimony was the Antimony King Mine in Nevada operated by Dowco Mining Company (Wyche, 1973).

For many years, NL Industries, Laredo, Texas, and the Sunshine Mining Company, Kellogg, Idaho, have been the major producers of antimony. Since

1973, however, United States Antimony Corporation has been converting antimony sulfide concentrates to metallic antimony at its Thompson Falls, Montana smelter. Annual capacity was estimated at 600 tons of the metal in 1973, but the company at that time anticipated increasing its output to 800 tons, and was weighing the possibility of producing antimony trioxide (Wyche, 1973).

Antimony is also recovered in antimonial lead from domestic lead ores at primary lead smelters. According to Wyche (1973), primary lead smelter production of antimony in 1973 was 17,206 tons. Of this, 16% was produced as metal, 65% as oxide, 7% as antimonial lead, 11% as ground residue, and 1% as sulfide.

In October 1975, Asarco Incorporated announced that it will build a $7-million, 1,825-ton/year antimony metal refinery at its El Paso plant in Texas, which is expected to produce 99.5% pure antimony by the end of 1976.

Other companies listed as producers or suppliers of antimony, include Atomergic Chemetals Co., New York; Richardson-Merrell, Inc., a subsidiary of J.T. Baker Chemical Co., Phillipsburg, New Jersey; and Hooker Chemical Corp., Niagara Falls N.Y.; Belmont Metals, Inc., a division of Belmont Smelting and Refining Works, Inc., Brooklyn, New York, offers jewelry casting alloys, prototypes, and precision castings of Britannia (an alloy chiefly of tin, copper and antimony).

Major producers of antimony oxide include NL Industries; Asarco, Inc.; Chemetron Corp., Chicago; Harshaw Chemical Co., Gloucester City, N.J.; McGean Chemical Co., Cleveland; and M & T Chemicals, Inc., Rahway, N.J. In early 1974, Associated Metals and Minerals went on stream in Texas City, Texas, with an antimony oxide plant having an initial capacity of 1.8 million lb/year.

Distributors of antimony oxide include:

- The Graymor Chemical Co., Clifton, N.J.
- Helm New York Chem. Corp., New York and Chicago
- ICC Solvent Chemical Sales Corp., New York
- Koch Int'l Chemical Co., Inc., Wellesley Hills, Mass.
- McKesson Chem. Co., San Francisco
- Morgan Chemicals, Inc., Williamsville, N.Y.
- Samincorp, Inc., New York
- Whittaker, Clark & Daniels, Inc., South Plainfield, N.J.

Antimony pentasulfide is manufactured by or is available from Atomergic Chemetals Co., New York; City Chemical Corp., Jersey City, N.J., and Great Western Inorganics, Inc., Golden, Colorado. Antimony trisulfide is manufactured by or is available from Atomergic Chemetals Co., New York; Barium & Chems, Inc., Steubenville, Ohio; Belmont Metals, Inc., a division of Belmont Smelting and Refining Works, Inc., Brooklyn, N.Y.; Chemetron Corp., Cleveland, Ohio; Hummel Chemical Co., Inc., South Plainfield, N.J.; McGean Chemical Co., Inc., Cleveland, Ohio; and Rare Metal Products Co., Atglen, Pa.

Manufacturers and distributors of antimony compounds available commercially are presented in Table V.

TABLE V

MANUFACTURERS AND SUPPLIERS OF ANTIMONY COMPOUNDS

COMPOUND	COMPANY	LOCATION
Antimony acetylacetonate	MacKenzie Chemical Works, Inc.	Central Islip, N.Y.
Antimony ammonium fluoride	City Chemical Corp.	Jersey City, N.J.
Antimony arsenate	City Chemical Corp.	Jersey City, N.J.
Antimony fluoborate	Harstan Chemical Corp.	Brooklyn, N.Y.
Antimony oxalate	City Chemical Corp.	Jersey City, N.J.
Antimony oxychloride	City Chemical Corp.	Jersey City, N.J.
Antimony oxysulfide	City Chemical Corp.	Jersey City, N.J.
Antimony pentachloride	American Hoechst Corp.	Somerville, N.J.
	Atomergic Chemetals Co.	New York, N.Y.
	Electronic Space Products, Inc.	Los Angeles, Calif.
	Great Western Inorganics, Inc.	Golden, Colo.
	Hooker Chemical Corp.	Niagara Falls, N.Y.
	Mallinckrodt Chemical Works	St. Louis, Mo.
	Richardson-Merrell, Inc.	Phillipsburg, N.J.
Antimony pentafluoride	Allied Chemical Corp.	Metropolis, Ill.
	American Hoechst Corp.	Somerville, N.J.
	Electronic Space Products, Inc.	Los Angeles, Calif.
	Great Western Inorganics, Inc.	Golden, Colo.
Antimony pentasulfide	City Chemical Corp.	Jersey City, N.J.
	Great Western Inorganics, Inc.	Golden, Colo.
Antimony pentoxide	American Hoechst Corp.	Somerville, N.J.
	Atomergic Chemetals Co.	New York, N.Y.
	Electronic Space Products, Inc.	Los Angeles, Calif.
	Great Western Inorganics, Inc.	Golden, Colo.
	Harshaw Chemical Co.	Gloucester City, N.J.

TABLE V (cont'd)

COMPOUND	COMPANY	LOCATION
Antimony potassium tartrate	*Aceto Chemical Co., Inc.	Flushing, N. Y.
	*American Firstoline	New York, N.Y.
	*Browning Chemical Corp.	New York, N.Y.
	City Chemical Corp.	Jersey City, N.J.
	*McKesson Chemical Co.	San Francisco, Calif.
	Pfizer, Inc.	New York, N.Y.
Antimony silico oxide	NL Industries	Philadelphia, Pa.
		St. Louis, Mo.
Antimony sulfate	Apache Chemicals, Inc.	Seward, Ill.
Antimony tetroxide	Apache Chemicals, Inc.	Seward, Ill.
	Atomergic Chemetals Co.	New York, N.Y.
	Cerac, Inc.	Menomonee Falls, Wis.
	Metalsmart	Great Neck, N.Y.
Antimony tribromide	Apache Chemicals, Inc.	Seward, Ill.
	Cerac, Inc.	Menomonee Falls, Wis.
	City Chemical Corp.	Jersey City, N.J.
	Great Western Inorganics, Inc.	Golden, Colo.
Antimony tri-n-butylate	Stauffer Chemical	Gallipolis Ferry, W. Va.
Antimony trichloride	Apache Chemicals, Inc.	Seward, Ill.
	Cerac, Inc.	Menomonee Falls, Wis.
	Chemetron Corp.	Cleveland, Ohio
	Electronic Space Products, Inc.	Los Angeles, Calif.
	Mallincrockdt Chemical Works	St. Louis, Mo.
	McGean Chemical Co., Inc.	Cleveland, Ohio
	Richardson-Merrell, Inc.	Phillipsburg, N.J.
	Stauffer Chemical Co.	Weston, Mich.
Antimony trifluoride	American Hoechst Corp.	Somerville, N.J.
	Apache Chemicals, Inc.	Seward, Ill.
	Atomergic Chemetals Co.	New York, N.Y.
	Cerac, Inc.	Menomonee Falls, Wis.
	Ozark-Mahoning Co.	Tulsa, Okla.

TABLE V (cont'd)

COMPOUND	COMPANY	LOCATION
Antimony triiodide	American Hoechst Corp.	Somerville, N.J.
	Apache Chemicals, Inc.	Seward, Ill.
	Electronic Space Products, Inc.	Los Angeles, Calif.
	Great Western Inorganics, Inc.	Golden, Colo.
Antimony trioxide	Asarco, Inc.	New York, N.Y.
	Associated Metals and Minerals Corp.	Texas City, Tex.
	Chemetron Corp.	Cleveland, Ohio
	Electronic Space Products, Inc.	Los Angeles, Calif.
	Harshaw Chemical Co.	Gloucester City, N.J.
	*Helm New York Chemical Corp.	New York, N.Y.
	*ICC Solvent Chemical Sales Corp	New York, N.Y.
	*Koch International Chemical Co., Inc.	Wellesley Hills, Mass.
	*Kraft Chemical Co.	Chicago, Ill.
	M & T Chemicals, Inc.	Baltimore, Md.
	McGean Chemical Co., Inc.	Cleveland, Ohio
	*McKesson Chemical Co.	San Francisco, Calif.
	NL Industries	Highstown, N.J.
	Richardson-Merrell, Inc.	Phillipsburg, N.J.
	*Samincorp, Inc.	New York, N.Y.
	U.S. Borax Chemical Corp	Los Angeles, Calif
	*Whittaker, Clark and Daniels, Inc.	South Plainfield, N.J.
Antimony trisulfide	Apache Chemicals, Inc.	Seward, Ill.
	Barium and Chemicals, Inc.	Steubenville, Ohio
	Belmont Metals, Inc.	Brooklyn, N.Y.
	Cerac, Inc.	Menomonee Falls, Wis.
	Chemetron Corp.	Cleveland, Ohio
	General Metallic Oxides Co.	Jersey City, N.J.
	Hummel Chemical Co., Inc.	South Plainfield, N.J.
	McGean Chemical Co., Inc.	Cleveland, Ohio
	Rare Metal Products Co.	Atglen, Pa.
Sodium antimonate	M&T Chemicals, Inc.	Baltimore, Md.
	Chemetron Corp.	Cleveland, Ohio

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* Supplier

3. Production Methods and Processes

a. Antimony

Antimony is extracted from its sulfide and oxide ores, or from mixed ores, by volatilization, smelting, and liquation -- the choice of method being governed by the antimony content of the ore used. For low-grade ores, carrying from 5 to 25% antimony, the volatilization process is used. Medium-grade ores, containing 25 to 45% antimony, are smelted in a reverberatory furnace or a blast furnace. High-grade ores, containing 45 to 60% antimony, are liquated. Reverberatory furnaces or blast furnaces are also used for the smelting of the products of liquation and volatilization, and for the smelting of concentrated antimony ores. In addition, some complex antimony ores are treated by electrolysis or by leaching and electrolysis to recover the antimony. These various manufacturing processes are described briefly below, although all are not used in the U.S.

NL Industries, one of the two U.S. major manufacturers, produces antimony metal and antimony trioxide at its Laredo, Texas smelter. Primary smelting of the metal is carried out in a blast furnace using ores imported principally from Mexico, although not exclusively as other ore sources from various parts of the world are used. For the most part, oxide-type ores are used in the process. After smelting, the metal is further refined and cast into ingots (Hornedo, 1976).

Since 1956, the other major U.S. producer, the Sunshine Mining Company Kellogg, Idaho, has recovered antimony from its Ag-Cu ores by leaching and electrolysis, yielding a metal with less than 0.05% impurity.

A reverberatory-type furnace is used in United States Antimony Corporation's refining plant, which converts antimony sulfide ore into metallic antimony, near Thompson Falls, Montana.

For its new plant in El Paso, Texas, Asarco will use one-third feed material (copper-antimony-silver concentrates) from the Coeur d'Alene mining district with the balance from their Galena Mine, both near Wallace, Idaho. For this plant Asarco's central research laboratories developed a new process that will employ a reverberatory-type furnace in which the concentrates will be melted and then tapped continuously from the furnace for granulation. The resulting granules will be dissolved and an electrochemical process will be utilized for the removal of antimony from solution. The remaining copper and other metals are to be sent to the plant's copper smelting section for processing into 99.5% pure anode copper (Asarco, 1975).

The Bunker Hill and Sullivan Mining and Concentrating Company, Idaho, uses the electrolysis process to recover antimony.

In a pre-smelting process, antimony ores are first concentrated by the flotation process. In this process, the ores are crushed and wet-ground to particles ranging from 65 to 200 mesh and the resultant slurry is conditioned with a collecting reagent (usually xanthates) and a frothing agent (usually pine oil or a long-chain alcohol) and fed to the flotation cells. In the cells, where air is added, antimony sulfide particles, attached to the air bubbles, are removed as a concentrate in the froth. The gangue, flowing through the flotation cells is removed as tailings. The flotation concentrate is thickened and filtered and shipped to a smelter for reduction to antimony metal.

In the antimony oxide volatilization process, low-grade (5 to 25% Sb) sulfide ores are roasted with coke (or charcoal) and air (controlled aeration) to form antimony trioxide.

$$Sb_2S_3 + 6O_2 \rightarrow Sb_2O_6 + 3SO_2$$

Furnaces used for the roasting are shaft type, rotary kiln, converters, or blast roasters. Heat from the sulfide ore converts and volatilizes the antimony sulfide to antimony trioxide, which is recovered in flues, condensing pipes, baghouses, or Cottrell precipitators, or combinations of these. The trioxide is generally impure and so is shipped (sometimes after briquetting) to a smelter, where it is reduced to antimony metal. In some instances, according to Kirk-Othmer (1963), a high-grade oxide, identified as Stibnox in the trade, results from the oxide volatilization process. This product requires a particular charge and special process conditions.

The reverberatory furnace smelting process handles medium-grade (25 to 40% Sb) antimony sulfide ores and flotation concentrates, antimony trioxide from the volatilization process, or antimony sulfide from the liquation process. In the furnace, the concentrates are mixed with charcoal, an alkaline flux (e.g., of soda, potash, and sodium sulfate) and recycled slag.

$$SbS_3 + 5O_2 \rightarrow Sb_2O_4 + 3SO_2$$

$$Sb_2O_4 + 4C \rightarrow 2Sb + 4CO$$

The sulfide ores are converted to oxide ores, which are reduced to metallic antimony. Flue gases are passed to baghouses or Cottrell precipitators to minimize the loss of antimony from the charge by volatilization and to recover the volatilized antimony oxides. The purpose of recycling the slag is to recover antimony from it. The molten antimony drawn off from this process is impure or crude antimony.

In the blast furnace smelting process, intermediate-grade (25 to 40% Sb) antimony sulfide ores and flotation concentrates, antimony oxide concentrates, liquation residues, mattes, rich slags, briquetted fines or flue dusts are processed with coke.

$$SbS_3 + 5O_2 \rightarrow Sb_2O_4 + 3SO_2$$

$$Sb_2O_4 + 4C \rightarrow 2Sb + 4CO$$

The furnaces, usually water-jacketed blast furnaces, use a high smelting column, low air pressure, and separation of slag and metal in a forehearth to reduce the charge to impure antimony metal, which is continuously removed. A large amount of slag is generated but it serves the purpose of reducing the loss of antimony by volatilization. This slag, generally containing less than 1% of antimony, is dumped.

In the liquation process, high-grade (45 to 60% Sb) antimony sulfide ore is

$$Sb_2S_3 + 6O_2 \rightarrow Sb_2O_6 + 3SO_2$$

heated to 550 to 600°C in a perforated pot to separate the antimony sulfide (crude antimony) from the gangue. The molten sulfide is tapped and collected in a lower container. When the heating is done in a reverberatory furnace, the molten antimony sulfide is removed continuously. Although this continuous collection method is more efficient, it involves the use of a reducing atmosphere that will prohibit loss by volatilization. Volatilized antimony oxides are recovered in baghouses or Cottrell precipitators. The residue from this process contains 12-30% antimony. The residue is passed to a volatilizing furnace for recovery of its antimony content as antimony oxides.

In the electrolysis process, impure metal is refined to an electrolytic antimony that is at least 99.9% pure. Anodes of crude antimony are hung on anode bars in an electrolytic cell alternately with starter cathodes of 99.9+ antimony hung on cathode bars. For best results, an electrolyte solution of antimony fluoride and sulfuric acid is used. The anodes dissolve into the electrolyte and most of the anode impurities are removed periodically as slimes from the bottom of the cell. The metallic antimony is plated out of the electrolyte onto the high-purity antimony cathodes.

Leaching and electrolysis is a method used to recover antimony from its complex ores. The ores are leached with a solution of sodium hydroxide.

$$4Sb_2S_3 + 8S + 18NaOH \rightarrow 5Na_3SbS_4 + 3NaSbO_3 + 9H_2O$$

The leaching may be done directly or the concentrates may be converted into a complex matte before leaching. The sodium thioantimonate leach solution is electrolyzed in a diaphragm cell using an iron or lead anode and an iron or mild-steel cathode. A 93 to 99% pure antimony is deposited on the cathode. One disadvantage of this process is that the electrolyte becomes contaminated with thiosulfate, sulfates, and other compounds, none of which are solvents for the antimony sulfide. The contaminated electrolyte must, therefore, be changed. It is either discarded or reduced to sodium sulfide, which can be reused as a leach liquor for antimony.

Primary antimony is also produced as a by-product of lead smelting and refining, if Sb is present in the Pb ores in a large enough quantity. Then, the dust emissions, drosses and slags resulting from the smelting and refining of these lead ores make the recovery of Sb feasible. This by-product is obtained as metallic antimony or antimonial lead, which can be processed to yield antimony oxide or sodium antimonate.

Undoubtedly, this is the method used by Asarco to produce its antimonial lead and antimony oxide. Although Asarco buys all types of ore concentrates from the world market, its galena mine in Idaho may be the prime source of Asarco's antimony concentrates.

Additionally, much of the antimony consumed in the U.S. is secondary antimony, which is recovered from antimony-containing scrap lead and tin, such as battery plates, type metal, bearing metal, antimonial lead, etc.

b. Antimony Compounds

Stibine (hydrogen antimonide, antimony trihydride), SbH_3, is formed by treating metal antimonides with acid, by the reduction of antimony compounds from high oxidation states, or by the electrolysis of acid or alkaline solutions using a metallic antimony cathode. In the classical synthesis, a 33% Sb-67% Zn alloy is dissolved by hydrochloric acid to form gaseous products containing up to 14% stibine. Stibine is also formed by the reduction of trivalent Sb in acidic aqueous solutions. According to Kirk-Othmer (1963), typical reactions include:

$$Zn_3Sb_2 + 6H_3O^+ \rightarrow 3Zn^{++} + 2SbH_3 + 6H_2O$$

$$SbO_3^{3-} + 9H_3O^+ + 3Zn \rightarrow SbH_3 + 3Zn^{2+} + 12H_3O$$

Antimony pentachloride can be prepared by the action of chlorine on antimony, or by the action of excess chlorine on the trichloride, thus deepening the color of antimony pentachloride. The excess chlorine is removed by a stream of dry carbon dioxide.

Antimony pentasulfide is made by converting the trisulfide to thioantimonate(V) by boiling with sulfur in alkaline solution. The resulting mixture is decomposed with hydrochloric acid and the pentasulfide is liberated.

Antimony pentoxide is an acidic oxide prepared by the action of concentrated nitric acid on the metal or the trioxide.

Antimony potassium tartrate is manufactured from potassium bitartrate and metallic antimony in the presence of nitric acid or from potassium bitartrate and solid antimony oxide.

Antimony trichloride is prepared by the reaction of chlorine on antimony.

Antimony trioxide is obtained during the smelting of antimony sulfide for the production of antimony,

$$2Sb_2S_3 + 9O_2 \rightarrow Sb_4O_6 + 6SO_2$$

It can also be prepared by combining antimony directly with air or oxygen,

$$Sb_4 + 3O_2 \rightarrow Sb_4O_6$$

Another method involves the alkaline hydrolysis of antimony trichloride, bromide, or iodide followed by dehydration of the hydrous oxide obtained (Kirk-Othmer, 1963).

Antimony trisulfide occurs in nature as black crystalline stibnite but can be manufactured in several ways: (1) by treating the Sb trichloride with hydrogen sulfide; (2) by treating the trichloride with sodium thiosulfate solution; (3) or by heating metallic antimony or the Sb trioxide with sulfur.

4. Market Price

The price of antimony from 1960-1975 (New York) is shown in Table VI. From a low of about 31¢/lb in 1960, Sb prices rose gradually through 1968 (when the price was almost 46¢/lb) to a high in 1970 of about $1.44/lb. Prices then dropped, only to rise sharply again in 1974. The rather stable trend culminating in the present high prices (which are trending downward) was interrupted by the peak prices in 1969 and 1970 which reflected tight supplies in 1969. Prices subsequently dropped with available imports.

The price of antimony metal and antimony oxide rises and falls due to several factors under the general heading of supply-demand, but with special inhibitors and stimuli. Part of the supply of antimony depends on activity in the refinement of other metals where antimony is (or may be) recovered as a by-product. If activity drops in these other metals markets, antimony residues would not be available for processing. In addition, imports are a significant factor in antimony production and the political conditions in these supply countries affect the U.S. supply (Semling, 1974).

In 1972, about 70% of the U.S. antimony oxide consumption went into flame retardant applications, principally plastics. Enforcement of the recently enacted Flammable Fabrics Act of 1973 is likely to contribute to the demand for antimony oxide (Chem. Wk., 1973). On the other hand, a substantial portion of the antimony oxide used in flame retardants is associated with the automotive plastics industry, which is currently at a depressed level. In this instance, antimony oxide is not being bought in proportion to its anticipated need (Chem. Mkt. Rept., 1975).

TABLE VI

ANTIMONY PRICE
(NY, Cents per pound)

	1960	31.30
	1961	33.89
	1962	34.75
	1963	34.75
	1964	42.22
	1965	45.75
	1966	45.75
	1967	45.75
	1968	45.75
	1969	57.57
	1970	144.19
	1971	71.18
	1972	59.00
	1973	68.50
	1974	181.76
(First Quarter)	1975	204.49
(Second Quarter)	1975	182.71
(Third Quarter)	1975	160.00

Source: Compiled from data in U.S. Bureau of Mines Minerals Yearbook, 1962-1973 and from the Division of Non-ferrous Metals, U.S. Bureau of Mines, Mineral Industry Surveys, December 18, 1975.

Therefore, producers and distributors who were stockpiling antimony oxide to meet the expected demand did not find a ready market. This has produced the price fluctuation in the first part of 1975, dropping from the highs of a year or two ago.

In the fall of 1974 the price of antimony oxide reached a peak price which was 180% over that one year earlier in 1973. Three months later, M & T and NL Industries had dropped their price 60¢, or 25% to make their product competitive with that of other suppliers (Chem. Mkt. Rept., 1975), and Chemetron followed suit three months later with a 20¢, or 10.8% drop (Chem. Mkt. Rept., 1975). Harshaw had initiated this price cutting in 1975 and McGean was expected to follow along with the rest. All these firms were citing a lower cost of raw materials; none suggested that the price had remained inflated from the previous strong-demand year 1973 (Chem. Mkt. Rept., 1975). With inventories being used up and with an anticipated 4% annual increase in demand to 1985, the price of antimony is expected to resume its upward climb.

5. Consumption and Market Trends

Forecasts of U.S. antimony demand, calculated from the U.S. twenty-year primary demand trend of 21,000 tons for 1973, indicate a probable average annual growth rate between 1973 and 2000 of 3.2 percent compared to 2.1 percent for the rest of the world. Over the same period secondary or "old scrap", antimony demand is expected to grow at an annual average of 2.6 percent in the U.S. compared to 2.0 percent for the rest of the world. Projections for end-use demand of antimony in the U.S. ranges between 65,000 and 112,000 short tons by 2000 as compared to 44,422 short tons in 1973 for such applications as transportation, fire retardants, rubber products, chemicals, ceramics and glass, machinery, etc. (Wyche, 1975a). However, because of the general economic downturn in the last two years, producers have been utilizing their stockpiled antimony. As these stockpiles are depleted, the increasing need will urge the production upwards (Wyche, 1973). Industrial stocks of primary antimony are shown in Table VII.

TABLE VII

INDUSTRY STOCKS OF PRIMARY ANTIMONY IN THE U.S.

(Short tons, antimony content)

							(Third Quarter)
Stocks	1969	1970	1971	1972	1973	1974	1975[2]
Ore and concentrate-----	2227	2973	3582	3562	5585	6275	11145
Metal-------------------	1273	1598	1367	1332	1540	809	1292
Oxide-------------------	2053	2932	2697	3179	2074	3732	3326
Sulfide-----------------	108	39	22	182	31	35	32
Residues and slags------	307	948	647	176	526	549	486
Antimonial lead[1]--------	371	357	322	191	322	294	521
TOTAL----------	6339	8847	8637	8622	10078	11694	16802

[1] Inventories from primary sources at primary lead refineries only.

[2] 1975 figures are estimated 100% coverage based on reports from respondents that held 90% of the total stocks of antimony at the end of 1974.

Source: Wyche, 1973 and 1975b.

Antimony metal and antimony oxide account for most of the tonnage, as shown in Table VIII. The principal uses for the metal are in antimonial lead, in bearings, and in ammunition. Industrial consumption of metal for these and other uses are summarized in Table IX. Most of the secondary production goes into lead battery grids and other hard-lead alloys.

In 1974, antimony consumption was affected by the downturn in the automotive market (Chem. Mkt. Rept., 1975) with a corresponding drop in use of antimony for batteries and in use of antimony oxide in flameproofing and flame retardants.

As previously mentioned, new laws and regulations concerning requirements for fire retardancy have influenced the antimony market and will continue to do so. In addition to the automotive industry, there will be a need for flame retardants in television cabinets, particularly those made of plastics. Also, fire laws pertaining to polyvinyl chloride, wallboard, plastic floor coverings and ceiling materials necessitate the use of flame retardants. Though product identification was diverse, about 70% of all antimony oxide sold (in 1972) was for the flame-retardant market (Chem. Wk., 1973). Despite attempts to find an alternative to antimony oxide as a flame retardant (for example, borate compounds and chlorinated paraffins), Finney (1975) reported that increased prices for substitute materials helped keep antimony oxide competitive. The potential effect of new U.S. regulations plus the established need for flame retardants in construction (plastics) and home (fabric and plastics) are expected to help stabilize the antimony market (Chem. Mkt. Rept., 1975).

It appears that the increase in flameproofing chemical usage in 1974 and the drop in ceramic, plastic, pigments, and other products may indicate a shift in reporting practice inasmuch as the use of antimony oxide in these other areas is usually related to flameproofing or fire resistant properties. The steadily declining use in type metal represents a major shift in the printing industry from letterpress to other methods.

TABLE VIII

INDUSTRIAL CONSUMPTION OF PRIMARY ANTIMONY

(Short tons antimony content)

	1969	1970	1971	1972	1973	1974	(Third Quarter) 1975[1]
Ore and Concentrate----	507	380	387	1226	582	1032	399
Metal------------------	6275	4989	5080	5473	5824	4362	3233
Oxide------------------	8756	7157	6944	8389	10970	9457	4806
Sulfide----------------	72	46	28	104	255	62	20
Residues---------------	330	384	136	201	1839	2066	552
Byproduct Antimonial lead-------	1903	981	1132	731	1143	1062	502
TOTAL-----------	17843	13937	13707	16124	20613	18041	9512

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[1] Inventories from primary sources at primary lead refineries only.

Source: Wyche 1973 and 1975b.

TABLE IX.

INDUSTRIAL CONSUMPTION BY PRODUCT

(Short tons antimony content)

Product	1969	1970	1971	1972	1973	1974	(Jan-Sept) 1975
Metal products:							
Ammunition	115	102	67	64	122	121	221
Antimonial lead[1]	6723	5246	5430	6149	8027	7251	3314
Bearing metal and bearings	758	481	515	559	527	476	233
Cable covering	55	38	36	19	12	16	23
Castings	33	16	20	39	65	31	1
Collapsible tubes and foil	56	35	22	20	12	18	7
Sheet and pipe	105	77	74	108	97	69	39
Solder	242	286	178	177	191	205	52
Type metal	541	220	177	142	134	107	37
Other	137	73	102	105	104	135	28
TOTAL METAL PRODUCTS	8765	6574	6621	7382	9291	8429	3955
Nonmetal products:							
Ammunition primers	37	27	23	23	18	11	8
Fireworks	30	17	4	4	5	11	4
Flameproofing chemicals and compounds	2096	1774	1524	2280	2906	4383	1617
Ceramics and glass	2108	1820	1840	1695	1917	1384	741
Pigments	722	610	592	644	644	460	142
Plastics	2558	1667	1810	2391	2920	1431	298
Rubber products	433	519	525	587	693	664	192
Other	1094	929	768	1118	2219	1268	415
TOTAL NON-METAL PRODUCTS	9078	7363	7086	8742	11322	9612	3417
GRAND TOTAL--	17843	13937	13707	16124	20613	18041	7372[1]

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1. Includes primary antimony content of antimonial lead produced at primary lead refineries.
2. 9512 short tons antimony content is estimated 100 percent coverage based on reports from respondents that consumed 80 percent of the total antimony in 1974

SOURCE: Wyche, 1973 and 1975b.

B. Uses

The applications to which antimony has been put are generally categorized as metal and non-metal products. Overall use of antimony by these product groups has been somewhat evenly balanced with the proportionally greater consumption shifting in the 1950's to the non-metal from the metal products group. In 1974, however, the total market declined and since then, figures show greater use in metallics. The U.S. industrial consumption of antimony in the manufacture of these metal and nonmetal products since 1969 is shown in Table VIII, p.36. In 1968, primary and secondary antimony (in terms of Sb content) were consumed in the following forms. Metal in solder, type metals, babbit metals, ammunition, storage batteries, etc. used 6,561 short tons; oxide in glass and clay products, rubber and plastics, pigments, textiles, chemicals, etc. used 9,363 short tons; sulfide in pigments and plastics used 75 short tons; antimonial lead (primary antimony) and antimonial lead and other alloys (secondary antimony) in storage batteries, cable covering, printing and publishing, communications, ammunition, etc. used 2,222 short tons and 23,699 short tons respectively.

1. Major Uses

a. Metals and Alloy Products

Antimony is not easily fabricated, primarily due to its extreme brittleness. As a result, it is rarely used as a pure metal, except perhaps in a decorative function. However, its hardness and stiffness make it an important alloying ingredient for metallurgical applications. The metal products in which primary antimony is used include antimonial lead and battery metal; bearing metals, bearings and type metal of antimony alloyed with tin or lead; Britannia or babbitt metal; pewter; ammunition, e.g., bullets; castings; sheet and pipe; cable covering; collapsible tubes and foils; solders (to add strength); etc. Very high-purity antimony has several semiconductor applications. When alloyed with aluminum, indium, gallium, cadmium, zinc, etc., it is used in infrared and Hall-effect devices, in diodes, and in thermoelectric piles and thermoelectric devices for cooling.

Antimony is used to blacken iron and coat metals. Antimony trichloride, in particular, has been used for bronzing iron, especially gun barrels, and for coloring zinc black. Together with nitrocellulose lacquers, antimony oxide (Sb_2O_3) yields films with excellent durability and chalking resistance. The favorable polishing properties of Sb_2O_3 add to its suitability for low-resin types of automotive finishes.

Antimony has been employed in metal finishing and electroplating. For example, antimony potassium tartrate (tartar emetic) has been used in electrolytic baths to deposit silver and antimony alloys on brass, copper and steel surfaces.

b. Non-Metal Uses

Antimony is used in such non-metallic products as fire retardants and flame-proofing chemicals; ceramics, enamels, and glass; plastics; pigments; rubber products; ammunition primers and fireworks; and in the manufacture of antimony compounds, such as antimony salts and antimonides.

(1) Flame Retardants for Plastics, Paint, Textiles, Rubbers and Paper

With the advent of regulations and requirements governing its applications, the selection of the optimum fire-retarding chemical additive is critical. The number and types of applications for antimony oxide compounds with flame resistant properties is vast. Various grades of antimony oxide are used for fire resistance in rubbers, plastics, textiles, paints and paper depending on specific needs and requirements. Specific products include:

Acrylonitrile - Butadiene - Styrene (ABS)
Alkyd coating compositions
Chlorinated rubbers
Elastomers
  - NR
  - SBR
  - Hypalon
  - Neoprene
  - ANB
Epoxies, e.g. epoxy printed circuits
Fiberfill for nightrobes, sleeping bags, quiltings and comforters
Polyesters
Polyethylene
Polyolefins
Polypropylene (PP)
Polystyrenes (general purpose and high impact polystyrene)
Polyurethane
Polyvinyl chloride, rigid and flexible (PVC)
Polyvinylidene chloride

Special plasticized resin compositions
Textile coatings and finishes

- nylons
- cottons
- polyesters
- tent fabrics
- tarpaulins
- industrial curtains
- canvas

The chemistry which leads to a flame retardant antimony oxide is based on the reaction of antimony and a halogen (usually chlorine) to form a halogenated compound like antimony trichloride at a temperature over 600°F. It is the antimony chloride which is flame retardant and may even be distilled into the substrate to give additional protection. The addition of about 3% antimony oxide to chlorine-containing polyester resins (with 25% chlorine) improves flame resistance. With lower chlorine content, more antimony oxide is necessary until the chlorine content is less than about 20%, at which point additional amounts of antimony oxide cannot compensate for deficiency in chlorine. (Robitschek and Bean, 1954)

Formulations for flame retarding vary considerably from material to material. Each application has a specific set of requirements necessary to meet the specific flame tests to which the products are subjected. The American Society for Testing of Materials, Underwriters' Laboratories, Inc. (UL), DOT, DOC, etc., all have special requirements, depending on the end use of the material, for which specific modifications must be made. In some cases flame retardants are applied to achieve a non-burning material; in other instances, a self-extinguishing feature is necessary.

For flame retardants in plastics, the effective level of antimony oxide (Sb_2O_3) used varies from as little as 2 to 5 parts per hundred parts to about 10% loading. In rubbers, the application of Sb_2O_3 may range from 5 to 30% levels of loading.

Treating fabrics for fire resistance is usually part of the finish and typically includes a fire, water, weather, mildew resistant (FWWMR) application. For textiles in roll form, the yard goods are run over rollers, down through a bath where the dry cloth becomes saturated with the special solution, and through a dryer for water and solvent removal. This is called a wet padding operation or water based treatment. Some flame retardant emulsions for textile coatings are applied by spraying or brushing them on the material being treated.

Antimony oxide is substituted for some of the pigment in the formulation of fire resistant paints. In the flame retarding of paper, Sb_2O_3 offers the advantage over other flame retardants of not being soluble in water.

(2) Other Uses in Paints, Textiles, Plastics, Rubbers and Paper

Due to its varying tinctorial strengths, antimony oxide is used as a mordant to achieve specific opacity ranges in paints and plastics. Antimony potassium tartrate is used as a mordant in fixing basic colors on cotton, leather and fur. Antimony potassium oxalate has been used in place of the tartrate as a mordant in fabric dyeing and printing. Antimony trichloride in solution is used as a mordant for patent leather and in the manufacture of furniture polishes. Antimony trifluoride, used as a mordant in dyeing, is usually in the form of double salts.

Antimony compounds used as pigments, primarily in paints, include antimony pentasulfide, antimony trisulfide and antimony trioxide. Commercial antimony oxide possesses valuable pigment properties and suppresses chalking tendencies of other white pigments. Although antimony oxide of the same volume has approximately half the opacity as rutile titanium dioxide, its staining power is one third that of titanium dioxide, which permits economies in using costly dyestuffs. (Paint Manufacture, 1964).

The incorporation of tartar emetic in vinyl chloride formulations assists in inhibiting and retarding discoloration. Similarly, it provides fastness to washing and light when used in textile printing.

The paint and plastics industries use Sb_2O_3 for its characteristic density and consequent covering ability or binding powers. Antimony pentasulfide is used in vulcanizing and coloring rubber.

Insoluble starch coatings for imparting water resistance to paper can be produced with antimony salts, particularly potassium pyroantimonate. Satisfactory results can be obtained if high temperatures are used, but the process is somewhat costly as are all other methods for making starch water-resistant (Casey, 1966).

(3) Uses in Glass

For glass, particularly optical glass, antimony oxide is used as a decolorizer and an antisolarant. During the decolorizing steps in glassmaking, the addition of sodium nitrate in combination with the Sb_2O_3 changes the antimony to the pentavalent state. The addition of Sb_2O_3 prevents the glass from changing color when exposed to the sun and imparts to the glass superior light-transmitting capabilities near the infrared end of the spectrum.

Antimony trisulfide is used in the manufacture of ruby glass. As a refining agent for optical glass and ruby red glass compositions, Sb_2O_3 assists in removing bubbles. It is a deodorizer in optical glass and a stabilizer in emerald green glass.

(4) Uses in Ceramics

Antimony oxide is an opacifying ingredient for porcelainized enamel. In addition, it contributes hardness and acid resistance to cast iron enamels.

Sprayed on the surface of red burning clay either as a water or oil suspension, Sb_2O_3 is used as a brick colorant. It is also used in ceramic pigments.

Antimony trifluoride is used in the manufacture of pottery and porcelains.

(5) Use in Pyrotechnics, Matches and Explosives

Antimony trisulfide and antimony pentasulfide are used in the manufacture of fireworks and matches. Antimony pentasulfide is also used in explosives and Bengal fires.

2. Minor Uses

a. Uses as a Catalyst or Chemical Agent

Antimony pentachloride is used as a chlorinating agent in organic synthesis, in dye manufacture, and as a catalyst in the preparation of some organic compounds. Antimony dichlorotrifluoride is used as a catalyst in the manufacture of organic fluorine compounds.

Antimony pentafluoride is a powerful oxidizing and fluorinating agent. The SbF_5-amine complexes are used as latent epoxy catalysts. In fluorosulfuric acid, SbF_5 forms a super acid system. These super acids are used in protonating weak acids, for stabilizing carbonium ions, for the preparation and investigation of new cationic inorganic species, and in fluorination reactions, according to Ozark-Mahoning Company's Special Chemicals Division.

Antimony oxide is used commercially as a catalyst in the production of polyester resins for fibers and films. Antimony trifluoride is used to catalyze fluorinations by HF and to manufacture chlorofluorides.

Antimony trichloride is used in making other antimony salts, in organic synthesis and as a catalyst. It is also a reagent for chloral, aromatic hydrocarbons, and vitamin A. In chemical microscopy, it is used for the identification of drugs by forming adducts and addition compounds.

Tri-n-butyl antimonite is a crosslinking agent for elastomerics, a catalyst for the production of polyesters, and an intermediate for other antimony compounds and catalysts.

b. Medicinal Uses

Antimony potassium tartrate is used medicinally as an emetic (tartar emetic) and in small doses as an expectorant in cough syrups. For the most part, treatment of parasitic infections with tartar emetic has been discontinued due to side effects attributed to its use. It is still used, however, in treating leishmaniasis and schistosomiasis. Because it is less toxic, antimony sodium thioglycollate has generally replaced tartar emetic as a medicinal. Antimony thioglycollamide has also been used instead of tartar emetic for the same reason, although it is more toxic than antimony sodium thioglycollate.

Although it also causes side effects, antimony sodium gluconate has been used in treating larva migrans. The trivalent compound is used as a schistosomicidal agent and the pentavalent compound as a leishmanicidal agent. Antimony sodium tartrate is also used for these purposes.

c. Veterinary Uses

Antimony barium tartrate was developed by the Zoological Division of the Bureau of Animal Industry, U.S. Department of Agriculture, and has been used for grapeworm infection in birds.

Antimony potassium tartrate is used in various dosages as an expectorant for the treatment of bronchitis in cattle, horses, sheep goats, swine and dogs. It is also used to treat ascariasis, leishmaniasis, trypanosomiasis and bilharziasis; as a ruminatoric in atony of cattle, sheep and goats, as an anthelmintic for horses; and for impaction of rumen in cattle.

Antimony trichloride has been employed as an escharotic in large-animal veterinary practice.

3. Chemical Reactions Involved in Uses

Antimony can be used as a catalyst to bring about the halogen exchange in the manufacture of chlorofluorohydrocarbons (Stephenson, 1966).

As a catalyst, antimony is usually used commercially in either the trivalent or pentavalent state. Pentavalent antimony is the more active catalyst but causes more side reactions. For this reason, trivalent antimony may be of greater use in the easier exchange reactions; pentavalent antimony may be used in particularly difficult reactions. A typical reaction proceeds as:

$$CCl_4 + SbF_3 \rightarrow CCl_3F + SbClF_2$$
$$SbClF_2 + HF \rightarrow HCl + SbF_3$$

with antimony the probable fluorinating agent. In the manufacture of fluorocarbons 11 and 12, the catalyst is SbF_3; for fluorocarbons 113 and 114, it is $SbCl_2F_3$.

4. Discontinued Uses

Consumption of antimony for galvanizing metal products and non-metal toy caps was reported for 1951 in the Minerals Yearbook of that date (Renick and Wright, 1954). However, neither appeared in the 1951 column in the comparable data in the Minerals Yearbook 1952 nor have they since. Galvanizing metal products were not reported prior to 1951 either, but non-metallic toy caps were previously included in other non-metal products.

Antimony potassium tartrate (tartar emetic) has been superseded by less toxic forms of antimony (e.g., antimony sodium thioglycollate, antimony thioglycollamide) for intravenous injections in treating tropical infections. It is no longer used in the control of insects as it formerly was.

5. Projected or Proposed Uses

A new low cost antimony stabilizer for the plastics industry, particularly for PVC multiscrew DWV and conduit compounds, was announced recently by Synthetic Products Company in Modern Plastics 52 (9), 147 (1975). Patented SYNPRON 1033 , this new antimony stabilizer is reputed to have cost performance advantages compared to organotin mercaptides at use levels of 0.2 to 0.5 phr.

Concern for product safety has brought about increased legislation and requirements for products with improved flame resistance. Some new underwriter's standards are indicative of this concern and bear directly on the increased use of flame retardants. For example, UL 492 is a standard for TV cabinets, which alone is expected to increase by some 1000 tons the demand for antimony trioxide (Sb_2O_3) in flame retardant chemicals (Mod. Plastics, 1975b).

Other developments show potential uses for antimony oxide. A smoke-inhibiting grade of antimony oxide is expected to be available in about a year from Chemetron Claremont Polychemical's ultrafine antimony oxide (at $2/lb).

6. Alternatives and Substitutes

Although antimony offers certain advantages in its many applications, it is not considered essential. In general, its use in paints, pigments and enamels can be substituted by mercury, titanium, lead, zinc, chromium, tin and zirconium. Alternative materials to antimony compounds have been used due to shortages in supply caused by military requirements. Certain economies in manufacture, (e.g., pigments, fire-retardants, lead-hardeners), have dictated replacing antimony with less expensive materials. In some cases, special organic compounds have been employed for flame proofing.

Alternatives for hardening lead with antimony include using tin, calcium and dispersion-hardened lead. Gould Inc. has substituted calcium for antimony in the lead alloys used for a heavy duty truck battery. Gould claims that the new battery will retain energy up to eight times longer than Sb-Pb batteries. Other advantages include consumption of less electrolyte, limited vent openings, and less loss of power during storage, according to Chem. Wk. 116 (19): 23 (7 May 1975).

Primarily for economic reasons, intumescent coatings may replace flame retardants in many products. The Underwriters' Laboratories (UL) has approved the application of intumescent coatings to the interior of

TV enclosures, thereby eliminating the need for modifying the cabinet plastic with flame retardants (Mod. Plastics, 1975b). For instance, lightweight magnesium oxychloride cement (Albi Duraspray), a product of Cities Service Company's Albi Manufacturing Corp., has been demonstrated as a thermal barrier when applied over exposed urethane foam (Mod. Plastics 1975a).

NL Industries' bimetallic smoke reducer, Ongard 1 (55¢/lb), can be substituted for some antimony oxide in PVC, PP, and urethane compounds. As an example, a 30% smoke reduction was achieved by using 3.5% of Ongard 1 and 3% of antimony oxide in DOP-plasticized calendering compounds. A 45% smoke reduction occurred when hydrated alumina replaced antimony oxide (Mod. Plastics, 1975b).

By partially or completely replacing antimony oxide with U.S. Borax FIREBRAKE® ZB(a refined zinc borate product) retention of translucency in fire-resistant reinforced plastic panels of halogenated polyesters is possible. A comparison was made between 5% concentration FIREBRAKE® ZB and 5% antimony oxide added to Hetron 93LS chlorinated polyester. Tests (ASTM D-1494) showed complete capacity with antimony oxide but 75% original light transmission with FIREBRAKE® ZB (Mod. Plastics, 1975b).

C. Potential Sources of Environmental Contamination and Control Practices

1. General

Included among the numerous organic and inorganic compounds, which man inhales daily from our polluted air are 23 trace metals. Nine of these are categorized as elements essential to life or health, seven are identified as nontoxic, and seven are metals with innate toxicities. Antimony is classified among the toxic metals, but is rarely detected in the air and, when it is, it is in minute concentrations, well below its threshold limit value(TLV) of 0.5 mg/m^3. Unlike cadmium, lead, mercury and nickel, antimony has not been categorized as a potential or actual public health hazard.

2. Production

Most recorded instances of antimony toxicity have been connected with its processing, handling, and use. The points of toxic exposure start with the extraction of antimony ores from their mineral deposits and include the subsequent beneficiation of the ores. Occupational exposure continues during the production of metallic antimony, antimony alloys, and antimony chemicals or intermediates, such as antimony oxide.

In the mining of antimony ores, mineral dust is released into the air from the drilling, blasting, crushing, and hauling operations. Antimony ores usually occur in the U.S. in vein type deposits in association with other metals (lead, zinc, copper, gold, silver). The deposits can be found at various depths and the underground mining procedures are those normally used for vein type deposits. The mining technology is similar to that used for hard rock minerals: drilling, blasting, loading and hauling the ore away from the working face for hoisting to the surface. During all of these procedures dust is liberated, but the highest concentration of dust probably occurs during the blasting of the mine face. However, since wet drilling is used and the mines are wet, the dust problem is not usually significant. Operating procedures keep men out of the mine when blasting is carried out and adequate ventilation is provided to remove any dusts generated.

Mineral ores are beneficiated to remove undesirable gangue and to increase their effective mineral content. Beneficiation usually involves crushing and grinding the ore to small particle size, flotation to separate the ore from the gangue, and roasting the separated ore to produce a product for smelting or refining operations. These processing steps, if they were performed in the dry state, would generate dust and, if they were not enclosed, the dust would be liberated to the ambient environment.

However, crushing is usually done in a closed air system, thus minimizing the amount of dust generated. Grinding and flotation are always carried out using water and hence are not a source of dust. In the roasting of ores, a flowing stream of air is used for combustion of the fuel. This air stream and the gaseous exhaust products of the roasting process capture the process-generated fine particles and dust and carry them to and through the exhaust stack of the roaster. Unless a proper dust collection or precipitation system is installed to contain these emissions, the dust will settle out of the exhaust plume and present a hazard to employees in that area. However, dust collecting systems are used.

Following the roasting operation, the product is transferred to a stockpile. During this transfer, as well as during transfer between operations, dust may be released to adjacent areas, unless it is properly contained. In the beneficiation process, poorly maintained equipment or improperly controlled processes provide opportunities for the release of potentially hazardous dust.

Antimony, because its oxides have high vapor pressure, can be beneficiated by volatilization. When lean antimony ores, even those with concentrations of 1 or 2% are heated, the antimony oxide volatilizes from the ore matrix. The fuming oxide condenses upon cooling and is collected in highly enriched condition. A properly maintained volatilizing furnace or collection system insures safe conditions. If, however, the furnace leaks and releases the fine oxide fumes to the ambient atmosphere, workers will be exposed to a potential hazardous situation. For safety purposes, during beneficiation local exhaust of working areas and direct exhaust of the roasting furnace are recommended.

In the production of antimony metal from its ores, the fossil fuel fired reverberatory furnace has been widely used. When the ore is charged to the smelting furnace with coke and an alkaline flux, a limited amount of antimony ore dust may be released. The charge is heated by the hot gas combustion product from open flames located at various points around the furnace hearth. Although at the start of the smelting operation the flow of hot gas in the furnace hearth entrains dust from the ore, the furnace is usually designed to carry this entrained dust to a gas cleaning system, where the dust is removed and collected before the gas is exhausted to the atmosphere. As smelting continues, the charge becomes molten. Then, antimony oxide fume is generated from the molten metal by oxidation of the metallic vapor, which is present above the surface of the bath. More fume is generated at the surface of the molten metal. This fume, like the dust from the charge, is carried by the furnace gas stream to the gas cleaning system for removal and collection. Because the by-product dust and fume are relatively rich in commercially valuable antimony oxide, the general practice is to maximize their collection and recovery from the exhaust gas stream. Again, leaks in the walls of poorly maintained furnaces will permit the antimony oxide dust or fume to escape into the work area. For the protection of the workers, recommended procedures include direct exhaust of the smelting furnace and local exhaust of working areas.

Another potential source of contamination arises when the smelter is tapped. During tapping, the ambient atmosphere in contact with the molten metal surface gives rise to antimony oxide fume, which is dispersed over the working area. Thus, workers near the smelter are exposed to the fume, unless the emissions are controlled by forced ventilation of the area to overhead canopy hoods that exhaust the fumes. If the molten metal tapped from the smelter is cast directly to pigs for remelting, the molten metal will not be in contact with the air and there will be one less possibility of fume release. If, however, the tapped metal goes to a ladle, a separate casting step is required. Again, the interaction of molten metal with the atmosphere will give rise to the release of antimony oxide fume at that location. In a further step, the crushing of cast antimony, there is less likelihood of significant dust release because the antimony does not shatter into fine particles. During the tapping, ladling, and casting operations, local exhaust of working areas is recommended.

Current regulations require the use of baghouses, electrostatic precipitators, and other devices to control air emissions of hazardous or potentially hazardous pollutants from smelters and other manufacturing plants. Effluent limitations recently proposed by the EPA for the ore mining and dressing industry suggest less than 0.5 of 1ppm Sb from antimony mines.

These regulations are contained in the Clean Air Act (as amended), the Federal Water Pollution Control Act (as amended), and the Marine Protection, Research and Sanctuaries Act (as amended). Consequently, most large U.S. industries are working to curtail the discharge of their hazardous pollutants into our air and water.

Air emissions, solid wastes and effluents generated by the two major producers of antimony in the U.S. can be characterized as follows. TRW (1973) assumed annual production in 1968 of 5,500 tons of antimony by the Laredo, Texas smelter of NL Industries. Although the smelter produced no significant amount of liquid effluent, it produced 2833.56 tons/year of airborne wastes, including NO (10.30 tons/year), SO_2 (60.5 tons/year), hydrocarbons (1.76 tons/year), CO (2.590 tons), and particulates (171 tons/year). Sixty-two lbs of particulates per ton of antimony were produced. The antimony content of the particulates was not known, but TRW assumed that it was 1%, and figured an airborne emission of approximately 28.1 g Sb/ton Sb produced. A further assumption (based on available data for the volume of stack gases produced as a function of the metal production in a lead smelter) provided an estimate that the Sb particulate emissions were 7.5 mg/m^3. If so, the emissions in 1972 were 15 times the accepted

Threshold Limit Value, but represented a decrease over previous emissions due to the installation in 1972 of four additional baghouses to collect the smelter emissions. In this same report, the slag or solid waste generated by the smelter is listed at 8,015 tons/ton Sb, or 44,000 tons annually. The slag, described as an insoluble mixture of metals and oxides, contains 1% Sb (60% metal, 40% oxides). NL Industries stores this slag outdoors on its property. The current soft antimony market precludes any immediate reworking of the slag.

In a recent discussion, a representative of NL Industries in Laredo confirmed that since 1972 the company had added the new baghouses, plus extra hoods and controls, and had succeeded in reducing the smelter emissions "tremendously" but not completely (Hornedo, 1975). In a later communication (Hornedo, 1976), NL Industries reported that its smelting and refining operations are equipped with high efficiency (99+%) fabric dust collectors. These collectors efficiently control particulate emissions, which currently have been found to be within acceptable limits and are not considered to be a problem. Blast furnace slag, containing approximately 1% antimony (of which 60% is in the metal form with the remainder in the form of silicates), is stock-piled in outdoor storage for future reclamation. There is no liquid effluent of any consequence emanating from the Laredo operations.

The Sunshine Mining Company, Kellogg, Idaho, in 1971 produced 854 short tons of antimony by electrowinning from the NaOH leach of their Ag-Cu ore. In 1971, the liquid effluent from this process contained 5 to 40 ppm Sb, but a recycling system scheduled for 1972 was expected to recapture the Sb in the effluent as soluble thioantimonate ion. As such it would be recycled to the main plant, and mixed with the antimony plant influent, the NaOH leach of Sunshine's Ag-Cu ore. Thus, the company planned to recover the 40 lb of Sb that it has been losing in its 9 to 12 tons/day of liquid effluent. With the installation of the recycling system, Sunshine expected to eliminate its settling ponds and stop the flow of 5 to 40 ppm of antimony pond effluent into the South Fork of the Coeur d'Alene river (TRW, 1973).

In a recent discussion, a representative of the Sunshine Mining Company reported that the company had installed the recycling system for its liquid effluent (Barr, 1975). Sunshine now discharges 900 gals/minute of effluent containing antimony in the range of 1.0-1.3 ppm (actual for the day of discussion was 1.26 ppm), which is a considerable decrease compared to the 5 to 40 ppm Sb discharged in 1971. However, Sunshine is still working to cut its antimony discharge in order to meet the EPA proposed antimony mine effluent limitation of less than one-half of 1 ppm Sb, and may have to go to acidification to do this. Because of the production method used, Sunshine has no air emission problem and generates no solid waste.

At this time, however, fish cannot survive in the river near the Sunshine operations. The company blames this condition on the sewage, dumped into the river by neighboring towns of 4-5000 population, which uses up all the available oxygen making it impossible for fish to live in the river. Before so much sewage was dumped into the river, the mine tailings apparently neutralized the sewage that found its way into the river, leaving enough dissolved oxygen for the fish to survive. In addition, certain fish came yearly to the area to spawn in the river. Fishery studies planned for 1976 are expected to make it possible for fish to return to the river. There is apparently no damage to vegetation in the area from the effluent discharged by the Sunshine Mining Company (Barr, 1975).

3. Transport and Storage

Table X summarizes current practices and procedures used by manufacturers in handling, storage and transport of antimony and its compounds. The information was culled from manufacturers' bulletins, materials safety data sheets, and technical brochures.

The potential exists for the release of dust or oxide fumes in manufacturing plants, when molten antimony metal or its oxide products are transported from one operating area to another, and in the transport of antimony and its compounds during their shipment to warehouses, to users and within the user's facilities. Cooper et al, 1968 reported that

TABLE X

CURRENT HANDLING PRACTICES

SUBSTANCE	COMPANY	STORAGE AND HANDLING	FIRE AND EXPLOSION HAZARD	EMERGENCY 1st AID	REACTIVITY DATA	SPILL OR LEAK PROCEDURES	SPECIAL PROTECTION INFORMATION SPECIAL PRECAUTIONS	WASTE DISPOSAL
Complex Antimony compound (Oncor 75RA) and Antimony Silico oxide (Oncor 23A)	NL Industries	External: Normal work day accumulations can be effectively cleansed with soap and water Change work clothes	No special fire hazards are anticipated but extinguishing media includes chemical, foam, CO_2, fog(water) on fires involving Oncor 75RA	Abnormal ingestion:* induce vomiting, give milk and magnesium sulfate (Epsom salts) Eyes: use first aid procedure for dust Consult physician if symptoms of illness *Call physician at once if antimony silico oxide is ingested	Hazardous decomposition products include: Sb_2O_4 Sb_2O_5	Sweep carefully, wear gloves, goggles, and respirator	Avoid dusting-use Bureau of Mines approved dust and/or fume respirator and goggles Ventilation as required to keep TLV below acceptable limits (for example, 25% Sb_2O_3 or 50% Sb_2O_4-0.5mg/m^3)	Landfill and smelter (Disposal must be done in accordance with local state and federal regulations)
High purity Sb	Atomergic Chemetals Co.	Very prone to surface contamination			Oxidizes readily when melted in the presence of air			Surface contamination may be removed by a short treatment with dilute aqua-regia (1:1) or dilute HNO_3 plus a small quantity of HF
Antimony trioxide (Antimony oxide) (Sb_2O_3)	American Smelting and Refining Co (Asarco)	Good personal sanitation must be practiced to avoid ingestion	Not flammable	Remove from exposure place at bed rest and call a physician in the event of acute symptoms during exposure	May generate Stibine under reducing conditions Stable	Sweep or shovel as much as possible into closed containers. Flush area with water spray. Wear approved respiratory equipment	Use Bureau of Mines approved respiratory protection, protective gloves and eye protection	Dispose of materials in closed containers. Be cognizant of potential water pollution problems
	J.T. Baker Chemical Co.	Wash thoroughly after handling		If swallowed, if conscious, induce vomiting by giving a tablespoon of salt in a glass of warm water. Repeat until vomit is clear. Give whites of eggs beaten with water. Keep patient warm and quiet. Call a physician	Ignites and burns when heated in air		Avoid breathing vapor or dust. Use respirator, local exhaust for ventilation, work in hood, wear rubber gloves, safety glasses and approved working clothes	According to local regulations

TABLE X

CURRENT HANDLING PRACTICES (Cont'd)

SUBSTANCE	COMPANY	STORAGE AND	FIRE AND EXPLOSION HAZARD	EMERGENCY 1st AID	REACTIVITY DATA	SPILL OR LEAK PROCEDURES	SPECIAL PROTECTION INFORMATION SPECIAL PRECAUTIONS	WASTE DISPOSAL
Antimony trioxide (Antimony oxide) (Sb_2O_3) (Cont'd)	Harshaw Chemical Co.	Avoid keeping or using food, tobacco and drink on the job site. Wash hands and face before eating or smoking. Vacuum clothing to remove dust. Change clothes daily or after exposure to heavy dusts. Shower in work area.				Sweep carefully to avoid causing large quantities of dust and place in container	Dusts should be collected and controlled at the suggested ventilation rate of 150 feet per minute air velocity Avoid ingestion	
	Chemetron Corp.	Standard package: 50 lb multiwall paper bag. Remove from the skin by using ample water and soap		If ingested, vomiting should be induced. Flush eyes with plenty of water for fifteen minutes			Use rubber gloves, safety glasses, and dust mask. Avoid inhalation	
Flowers of Antimony Sb_2O_3	McGean Chemical Co.	Normal handling and storage		If swallowed, induce vomiting. Call a physician Flush eyes with water. Call a physician Wash skin with soap and water	Stable	Sweep or vacuum up	Use dust mask, local exhaust ventilation, rubber gloves, safety glasses	Bury or return to manufacturer
Colloidal Antimony oxide	Nyacol Inc.	Grades with 10-30% concentration are in 55-gallon drums and should not be subjected to temperatures below 32 F, as precipitation may result in non-redispersible material Grades with 50% concentration are available in 30-gallon drums and can be frozen without harm May be readily sprayed, sponged or roller coated		If eye contact, wash out thoroughly with large amounts of water If skin contact, wash thoroughly with soap and water			In instances where drying or dusting may occur, wear protective clothing with a respirator Shower daily and wear fresh work clothes	

TABLE X

CURRENT HANDLING PRACTICES (Cont'd)

SUBSTANCE	COMPANY	STORAGE AND HANDLING	FIRE AND EXPLOSION HAZARD	EMERGENCY 1st AID	REACTIVITY DATA	SPILL OR LEAK PROCEDURES	SPECIAL PROTECTION INFORMATION SPECIAL PRECAUTIONS	WASTE DISPOSAL
Antimony trichloride (Anhydrous) ($SbCl_3$)	Manufacturing Chemists Association (MCA)	Ventilate for drum unloading Contain in metal drums and pails, single trip, full removable head, 2 and 5 gallon capacity Box in glass carboys-5 gallon capacity, pharmaceutical grade Keep dry and cool, protected from rain and sunshine Store in tightly closed containers out of contact with moisture	Nonflammable For fighting fires use suitable self-contained breathing apparatus	Skin contact and Mucous Membranes Flush with large quantities of water and wash with soap and water, remove contaminated clothing and wash Eye contact Wash with copious quantities of water for at least 15 minutes See physician at once Internal contact Induce vomiting with warm salt water See physician at once	In the presence of moisture, reacts to release hydrogen chloride gas	Shovel or sweep and flush with water (preferably hot) Brush clothing off, remove clothing and wash in water	Protect equipment and steel work in exposure area with acid resistant paint Handle in closed system to avoid atmospheric moisture or dehumidify room air. Use local exhaust or fume hood and ventilate work and storage areas Eye protection: Use chemical or special-type safety goggles and face shields Respiratory protection: Use industrial canister type gas masks approved by U.S. Bureau of Mines or cartridge respirators Head protection: Use safety or "hard" hat where appropriate otherwise brimmed felt hat Foot protection: Leather or rubber safety shoes with built in steel toe caps recommended Body, skin and hand protection: Use rubber aprons, long rubber gloves, sleeves of non-porous protective material Removal of all contaminated clothing immediately and clean before reuse Wash thoroughly	Dispose of small amounts of scrap or waste by flushing with hot water Seal large amounts of residues in containers and bury

TABLE X

CURRENT HANDLING PRACTICES (Cont'd)

SUBSTANCE	COMPANY	STORAGE AND HANDLING	FIRE AND EXPLOSION HAZARD	EMERGENCY 1st AID	REACTIVITY DATA	SPILL OR LEAK PROCEDURES	SPECIAL PROTECTION INFORMATION SPECIAL PRECAUTIONS	WASTE DISPOSAL
Antimony trichloride ($SbCl_3$) and Antimony pentachloride ($SbCl_5$)	J.T. Baker Chemical Co.	Keep in tightly closed containers in a dry place Do not get in eyes, on skin, or on clothing		Internal: If conscious, give plenty of water, induce vomiting until vomit fluid is clear. Give milk or egg whites beaten with water Keep warm and quiet External: Immediately flush area with plenty of water for at least 15 minutes while removing contaminated clothing If inhaled: Move patient to fresh air. Give artificial respiration if breathing has stopped Call a physician	Stable	If spilled, dissolve in minimum hydrochloric acid (concentrated reagent) Dilute with water until white precipitates form Add just enough 6M-HCl to redissolve. Saturate with hydrogen sulfide. Filter wash the precipitate, dry and package	Do not breathe vapors Use respirator, local exhaust for ventilation, and work in fume hood Wear protective rubber gloves, safety glasses, and approved working clothes	May be disposed by contacting an approved outside waste disposal service and/or according to local regulations
Antimony pentachloride (Antimony perchloride) ($SbCl_5$)	Hooker Chemical Corp.	Do not open unless properly instructed Do not clean or reuse container Shipped in 10-gallon (non-returnable) steel drum		(As above under external)	Do not add water to contents while in a container because of violent reaction	In case of spillage, flood carefully with large volume of water and provide adequate ventilation	Wash clothing before re-use	
Tartar Emetic (Antimony Potassium Tartrate)	Pfizer Chemicals Division		If involved in fire, water may be used	External Flush skin with water. If eye contact, flush with water and seek medical attention Internal: Call physician immediately Use plain, or soapy water or water with table salt or milk (3-4 glasses) to provoke vomiting	Stable	Remove by scoop or shovel	Use approved dust respirator. Use dust exhaust system at point of use for ventilation Protect with rubber or plastic coated gloves; chemical goggles; tightly woven, close-fitting clothes for minimum exposure of skin to dust and frequent showers	Wash down and flush to safe area

TABLE X

CURRENT HANDLING PRACTICES (Cont'd)

SUBSTANCE	COMPANY	STORAGE AND HANDLING	FIRE AND EXPLOSION HAZARD	EMERGENCY 1st AID	REACTIVITY D DATA	SPILL OR LEAK PROCEDURES	SPECIAL PROTECTION INFORMATION SPECIAL PRECAUTIONS	WASTE DISPOSAL
Tri-n-butyl antimonite (Antimony tri-n-butylate) (TNBA)	Stauffer Chemical Co.	Package and store under nitrogen(or an inert atmosphere) Store in carbon steel or brass cylinders of the 55 gallon drum 16 gauge size. Small quantities can be shipped in glass or high density polyethylene containers Remove and launder clothes before reuse	Flammable To fight fires use fog, CO_2 or dry powder	Ingestion: give large amounts of warm or salty water to induce vomiting, until vomitus is clear. Milk, eggs or olive oil may be given to sooth following vomiting Consult a physician if abdominal discomfort persists Eye contact: flush eyes immediately with large quantities of running water for a minimum of 15 minutes Consult a physician if irritation persists Skin contact: flush affected area with water Consult a physician if irritation persists Inhalation: Remove from contaminated atmosphere See a physician if respiratory discomfort persists	Extremely air and moisture sensitive Reacts rapidly with water, acids and bases to liberate antimony oxide and butyl alcohol Product of hydrolysis is very toxic		Avoid contact with skin, eyes and clothing Eye wash stations, approved safety glasses and approved chemical rubber gloves are recommended	Contact the Specialty Chemical Division for recommendations on unused material

concentrations of antimony in air, monitored in 1966 at 36 antimony processing plant locations under varied environmental conditions, ranged from 0.081 to 75 mg/m^3. Concentrations as high as 138 mg/m^3 were found in the area of the bagging operations.

In addition, the dust or vapor, when exposed to heat or flame, is a potential fire hazard. The dust, when exposed to flame, can be an explosion hazard. Conditions contributing to the instability of antimony compounds depend upon the specific compound of antimony involved. No generalization can be made, but the reactivity of these substances is discussed in more detail in Section IV.C.2, which covers current transport practices. Likewise, the incompatibility of antimony and its compounds, which also depends on the specific compound involved, governs transport practices and is discussed in Section IV.C.2. As an example, antimony metal in contact with nascent hydrogen will react to form stibine (antimony hydride), an extremely toxic compound.

Antimony and its compounds should be stored in dry, well-ventilated areas away from rain (or other water), heat and sunlight. Dust and fumes from leaky containers again are potential health, fire or explosive hazards. Fire extinguishers are recommended for storage areas and employees should not eat or smoke in these areas. Specific storage precautions are discussed in Section IV.C.2.

4. Uses

In addition to the contamination potentials that exist during the mining, production, transport, and storage of antimony and its compounds, a similar potential exists in those industries where antimony and its compounds are used in the preparation of other products or exist as byproducts of other operations. Exposure can be by inhalation, skin contact, or ingestion. Proper ventilation of the work areas, protective clothing protective respiratory devices, and other protective equipment should be used, depending on the specific compound involved. Individual procedures are discussed in Section IV.B and IV.C.1.

Some potential inhalation exposures occur during the production of lead/antimony alloys (which include pewter, Britannia metal, type metal, babbitt, and white metal) when antimony fumes and dust may be released. During the welding of metal products of alloys containing antimony (which include bearings, pipes, machine parts, and ornamental casting solder) antimony oxide fume may be released. Antimony dust is a potential hazard during the machining, grinding, buffing, and polishing of antimony-containing metal products. The oxide fume and dust may also be released during the casting, parting, and cleaning of battery grids and plates made of lead/antimony alloy. The liberation of toxic stibine from storage batteries may occur when nascent hydrogen reacts, in an acid medium, with antimony present in the battery.

In various type-setting operations (linotype, monotype, sterotype), antimony oxide vapors and dust may form. When antimony oxides are added to paints, pigments, enamels and glasses, there is a chance that the trioxide dust may form. In the manufacture of ceramics and glass, some antimony pentoxide dust may be formed. Skin contact with antimony trichloride solutions is a potential hazard during the dyeing and flameproofing of textiles, while skin contact with antimony pentachloride may occur during the manufacture of steel. Skin contact with and inhalation of sulfide dust may result from rubber compounding operations.

Antimony trioxide is used primarily as a fireproofing additive in plastics, textiles, and other products. During the blending and molding of flame retardant plastics, for example, there is the potential for environmental contamination.

In blending, dusting of the antimony oxide may cause direct atmospheric contamination. In addition, since occasional washing down of the dusty walls, floors, etc. is standard housekeeping practice, a smaller amount of antimony oxide may find its way into the waste water drains. In the molding of the flame retardant resins to produce finished goods, the materials are commonly exposed to temperatures as high as 250°C. At these temperatures, antimony oxide combines with the halogens in the resin to form volatile antimony species such as antimony oxyhalide and antimony trihalide. These materials typically condense on the cooler surfaces of the molds and ultimately must be removed by repolishing the mold surfaces.

Because of the high boiling points of these antimony halides, it is unlikely that a significant vapor concentration of these materials will be present at room temperature. Hence, the possibility of atmospheric contamination is remote.

Because very little of the antimony trioxide is used in the finished product (less than 2 to 3 parts/100 lb of plastics), it is disposed of as part of the plastic material. Antimony trioxide is discarded into municipal systems. There is no evidence that it is a harmful pollutant.

5. Disposal

Waste disposal methods for antimony and its compounds are governed now by local, state, and Federal ordinances. Permissible disposal methods depend upon the type of antimony compound involved. Solubility in water is an important factor.

Methods of waste disposal currently are landfill and smelter. Landfill carries with it the potential for hazards due to the leaching of trace metal into ground or surface waters. Smelting implies the possible emission of trace metals, which can be inhaled by people in the vicinity, or carried by the wind and deposited on soil or in waters. Because all trace metals can be toxic in high enough doses, and because antimony is identified as a trace metal with innate toxicity, its inhalation in high concentrations could be a health hazard. Ocean dumping of antimony waste has been practiced but ocean dumping of hazardous chemicals is under fire now because of potential harmful effects on marine life and on the marine ecosystem.

In mid-1974, five Gulf States protested EPA's granting of a permit to DuPont to dump waste chemicals, including antimony compounds in aqueous solutions or suspensions, in the Gulf of Mexico, some 230 miles south of Pensacola, Florida. Wastes, from DuPont's plant near Belle, W. Virginia, have been barged to Weswego, La., and dumped into the Gulf since 1969. The complainants emphasized the toxicity of antimony, which is used as a catalyst in the production of polyesters and is retained in the spent glycol recovered at the Belle plant. DuPont anticipates storing and handling the questioned waste in its own facilities by mid-1977. If the company cannot continue ocean dumping, storage and handling costs are estimated at \$10-\$13 million (Chem. Eng. News, 1974).

As a result of current regulations, land disposal sites are used more frequently for the disposal of sludges, slurries, and concentrated liquids. Recommended methods for disposal of antimony compounds include disposal in a chemical waste landfill, chemical pre-treatment before disposal by land burial, and encapsulation followed by disposal in a chemical waste landfill.

Chemical waste landfill can be used for the disposal of antimony pentasulfide, antimony sulfate, and antimony trisulfide. Chemical waste landfill differs from conventional sanitary landfill in that it requires the segregation of the wastes, the use of barriers to prevent leaching of the wastes to groundwater and, in some instances, the pretreatment (neutralization, chemical fixation, encapsulation, etc.) of the wastes.

In its 1974 Report to Congress on the Disposal of Hazardous Wastes (EPA Publication SW-115), the U.S. EPA Office of Solid Waste Management included antimony pentafluoride and antimony trifluoride in a list of nonradioactive high hazard waste compounds. Compounds so classified are considered unacceptable for normal disposal methods because of their potential for causing adverse health or environmental effects.

The following method is recommended for the disposal of antimony pentafluoride and antimony trifluoride. Dissolve the compound in dilute HCl and saturate with H_2S. Filter, wash, and dry the antimony sulfide precipitate. Air strip the dissolved H_2S from the filtrate and pass the filtrate into an incineration device equipped with a lime scrubber. After the stripped filtrate has reacted with excess lime, dispose of the precipitate (CaF-CaCl mixture) by land burial.

Nickel antimonide should be encapsulated before it is disposed in a chemical waste landfill.

NL Industries, Inc., which lists the decomposition products of antimony trioxide as Sb_2O_4 and Sb_2O_5, disposes its oxide waste by landfill and smelter; Asarco advises disposing waste trioxide in closed containers, being cognizant of water pollution problems.

To dispose of its antimony trichloride wastes, J.T. Baker Chemical Co. contacts an approved outside waste disposal service.

Antimony pentachloride can be decomposed by water, forming toxic, corosive hydrogen chloride. Hooker Chemical Co. decomposes its waste antimony pentachloride by feeding it at a controlled rate into water in a retention area, and adjusts its pH with lime or caustic before discharging the effluent.

In what may be a procedure for handling spills, Pfizer Chemicals Division reports that its antimony potassium tartrate (solubility in water = 8.7 g/100 ml) should be washed down and flushed to safe area.

On p.258 of its "Proposed Criteria for Water Quality," Vol. I, Oct. 1973, the EPA set the maximum acceptable concentration of antimony in marine or estuarine waters at 1/50 (0.02) of the 96-hour LC_{50} value determined, using the receiving water and the most important sensitive species in the locality as the test organism. Maximum allowable concentration of antimony in marine or estuarine waters is 0.2 mg/l. Insufficient data were available at that time to recommend a level that would present minimal risk of deleterious effects.

However, on p.4, Vol. II, of that same publication, the EPA reports that antimony in industrial effluents is rapidly removed by precipitation and adsorption, and no antimony was detected in the principal rivers of the U.S. in 1958-59. However, later information, as mentioned previously, indicates the presence of antimony in the sediments of waters in manufacturing areas.

6. Potential Antimony Release from Other Products and Industrial Processes

A large copper smelter near Tacoma, Washington, operating since 1890, produces as a by-product most of the arsenic trioxide used in the U.S. In addition to the arsenic that it releases into the environment, this smelter releases antimony in three ways: (1) as a stack gas into the air, 2×10^4 kg/yr of antimony oxides; (2) as antimony species in liquid effluent discharged directly into Puget Sound, 2×10^3 kg Sb/yr; (3) as crystalline slag particles dumped directly into the Sound, 1.5×10^6 kg Sb/yr. (Puget Sound also receives 250 kg of antimony/yr from sewage treatment plants).

In a recent study of the hazardous waste generated by seven industrial inorganic chemical industries -- chlorine, titanium dioxide (manufactured by the chloride process), chrome colors, mineral acids, aluminum compounds, potassium and sodium compounds, and industrial inorganic compounds (not elsewhere categorized) -- antimony was included as a hazardous constituent only in the waste streams generated by the production of titanium dioxide (chloride process) (Morekas, 1975).

Waste streams from the production of titanium dioxide by the chloride process contain a number of potentially hazardous constituents: antimony, arsenic, cadmium, chromium, cyanide, lead, mercury, and zinc. The waste discharge from one such titanium dioxide plant with an average wastewater discharge of 112 millions of liters/day contains 300 kg of Sb/metric ton of product. The raw material feed for this plant is over 95% TiO_2 grades of rutile and upgraded ilmenite (EPA, 1974).

Because many smelting and high-temperature combustion operations emit particles containing antimony and other toxic elements into the air, Davison et al (1974) measured the concentrations of 25 elements in the fly ash from a coal-fired power plant. The concentrations of antimony and nine other elements increased markedly with decreasing particle size, and in urban aerosols antimony is reported to have an equivalent mass median diameter of the order of 1 μm or less. Elements with their mass concentrated in the particle size range 0.5-10.0 μm are inhaled and deposited in the human respiratory tract.

In a study by the EPA National Fuels Surveillance Network, trace elements in gasolines and commercial gasoline additives contained a number of elements, including antimony. Concentrations of antimony in premium gasoline ranged from <0.003-0.05; in regular gasoline, <0.007-0.5; and in low lead gasoline were <0.10 μg/ml. In the additives, antimony concentrations ranged from <0.0005-0.0041 μg/ml. The presence of trace metals in gasoline is of environmental concern, since some have suspected biological toxicity, and they can be disseminated widely in the respirable range, at ground level. Antimony, however, is not included in the list of metallic elements (Ce, As, V, Ni, and Cr) cited as being suspect of biological toxicity (Jungers et al, 1975).

In mid-1971, several industrial companies, e.g., Dow Chemical, BASF Wyandotte, Georgia-Pacific, Kaiser Aluminum and Chemical, and Weyerhaeuser, were reported to have started company surveys to identify trace element pollutants likely to be involved in their processes. Antimony was one of 30 elements listed by Dow. On the basis of preliminary survey results, Dow eliminated some metal compounds from its processes and made some process and catalyst changes. Antimony, arsenic, beryllium, cadmium, and lead have caused industrial deaths.

According to Dr. H.A. Schroeder of the Dartmouth medical school's trace element laboratory, no metal is degradable and toxic metals in the environment pose a serious health hazard. He recommends the elimination of cadmium by controlling zinc smelting emissions, and urges the control of antimony and beryllium in the air by the reduction of particulate emissions from coal smokes. The biggest, but not the sole, source of atmospheric trace elements if probably the burning of fossil fuels, including gasoline with additives (Chem. Eng. News, 1971).

7. Chemical Reactions in the Environment

a. Hydrolysis

Because metallic antimony is a stable element, it does not readily react with moisture. Stibine (SbH_3) may be formed, however, by the hydrolysis of hydrochloric acid on Zn_3Sb_2or other metal antimonides.

In the presence of moisture the normal salts hydrolyze readily. Antimony trichloride releases hydrogen chloride gas, which represents a hazard in itself. Other antimony halides behave similarly. Antimony pentachloride reacts with water in a 1:1 molar ratio to form $SbCl_5.H_2O$ and in a 4:1 molar ratio to form $SbCl_5.4H_2O$. Further dilution gives HCl and hydrated (V) antimony oxide.

Although there is no direct evidence that antimonic (III) acid is a definite compound, antimony (III) oxide forms hydrates of indefinite composition believed to be related to the hypothetical antimonic (III) acid.

With water, antimony pentafluoride forms a clear solution from which $SbF_2.2H_2O$ can be crystallized.

Antimony tri-n-butylate reacts rapidly with water (and with acids and bases) to liberate antimony oxide and butyl alcohol. The combined products of hydrolysis are very toxic.

b. Oxidation

Metallic antimony, because it is stable or inert, is not readily attacked by air. Under controlled conditions, it will combine directly with air or oxygen to form its oxides, Sb_2O_3 and Sb_2O_5. A tetroxide, Sb_2O_4, a stoichiometric compound ($Sb_2O_3.Sb_2O_5$), is also formed.

Although antimony does not combine directly with hydrogen, antimony metal reacts with oxidizing materials and acids to produce stibine (SbH_3).

Antimony reacts with nitric acid to form hydrated antimony pentoxide, a gelatinous precipitate. An oxysulfate (undefined composition) results when antimony reacts with sulfuric acid.

The reaction of antimony with hydrochloric acid is slow unless air is present.

Oxiding agents react freely with stibine. Stibine, ignited in the presence of air or oxygen, forms water and antimony trioxide. Both metallic antimony and antimony trioxide will react with strong oxidizing agents to form hydrated antimony pentoxide, a gelatinous precipitate sometimes called antimonic acid.

In the absence of air, antimony trisulfide dissolves in alkaline sulfide solutions to form the thioantimonate (III) ion. This ion is oxidized in air to the thioantimonate (V) ion.

c. Photochemistry

In the presence of light and at 100°C, stibine reacts with sulfur to form antimony (III sulfide; with selenium to form antimony (III) selenide.

Photochemical properties of antimony trioxide have been investigated by Markham _et al_ (1958), who reported darkening of Sb_2O_3 and the production of peroxide under ultraviolet light. The reactions compared with those of zinc oxide. (Although this property of zinc oxide finds use in some copying papers, no equivalent use has been reported for the antimony compounds.)

d. Other

Antimony reacts with chlorine to form antimony tri- and pentachloride, and with sulfur to form antimony tri- and pentasulfide.

Some insoluble antimony compounds react with hydrofluoric acid to form fluorides or fluocomplexes.

D. Analytical Methods

1. Laboratory Standard Analytical Methods

Traditional analytical methods are available for determining the antimony concentration in ores, minerals, and even rafter dusts, but the greater precision and sensitivity offered by modern instrumental methods is required for the trace analysis of this material and its compounds in air, water, and in biological specimens. Colorimetric analysis of antimony has been employed widely for many years with the tetraethylrhodamine (Rhodamine B) method cited most frequently. The Rhodamine B test has a sensitivity of 6γ. In more recent years, such analytical techniques as atomic absorption spectrophotometry (a.a.s.) and neutron activation analysis (n.a.a.) have come into greater use.

In its Official Methods of Analysis for 1975, the Association of Official Analytical Chemists (AOAC) describes the determination of pentavalent Sb as residue in foods by means of an American Conference of Governmental Industrial Hygienists (ACGIH) - AOAC method. In an aqueous hydrochloric acid solution, antimony reacts with tetraethylrhodamine (Rhodamine B) and forms a colored complex which can be extracted with organic solvents such as benzene or toluene. The color intensity is measured at 565 nm spectrophotometrically. This method resulted from the ACGIH-sponsored collaborative tests on the Rhodamine B colorimetric method for antimony which the AOAC (then the Association of Official Agricultural Chemists) reported (Bartlett and Monkman, 1964).

The AOAC also reports the development of an atomic absorption method for antimony in the food additive titanium dioxide (Hoffman, 1969). This method is based on fusion with potassium hydrogen sulfate and extraction with methyl isobutyl ketone.

Atomic absorption spectroscopy is widely used in determining the presence of antimony. Data gathered with the Perkin-Elmer Model 303 instrument at 2175 Å wavelength with a 7 Å slit show the detection limit of 0.2 μg/ml with a sensitivity of 1 μg/ml/6%.

Determinations of antimony in air-acetylene flame show sensitivity at three wavelengths: 0.5 µg/ml/1% at both 2068.4 Å and 2175.9 Å and 1.2 µg/ml/1% at 2311.5 Å. The 2175.9 line is the most commonly recommended wavelength for antimony.

A similarity in sensitivity and detection limits for a.a.s. and atomic flame spectroscopy (a.f.s.) for antimony is shown (Norris and West, 1974) in various flames. The argon-hydrogen flame proved slightly superior to the air-hydrogen flame in each instance, though interferences were greater in the argon-hydrogen flame. In an atomic absorption spectroscopy study on the determination of antimony utilizing the generation of covalent hydrides (Thompson, Thomerson, 1974) data was obtained using a Shandon Southern Instruments A3400 atomic-absorption spectrophotometer. A 1 ml sample of acidified Sb^{III} treated with 2 ml sodium borohydride (1%) gave a detection limit of 0.0005 (2σ)/µg/ml at characteristic concentration at 1% absorption of 0.00061 µg/ml.

Flameless a.a.s. of antimony in metallurgical samples with a carbon rod atomizer requires solvent extraction of the chloride complex from HCl solution because of matrix effects of antimony adsorption (Yanagisawa et al, 1973).

ASTM Tests

The American Society for Testing and Materials (ASTM) gives a standard method for chemical analysis of antimony metal (E86-57) with a composition of 99 to 99.9% antimony, where the antimony is determined by difference.

In ASTM E87-58, a photometric method for chemical analysis of antimony metal is described. The sample is dissolved in a hydrobromic acid and bromine mixture, distilled, and treated with perchloric acid. A portion of the treated distillate is transferred to an absorption cell for photometric analysis.

Other ASTM standards describe methods for the chemical analysis of antimony depending on where it is found:

- For the detection of antimony in copper (ASTM 54-72) and in brasses (ASTM 36-45), the distillation-iodimetric method is used;
- In copper and copper-base alloys (ASTM-62-72), the method is iodoantimonite (photometric).
- In lead-base and tin-base solder metal bearing alloys (ASTM E57-60) the standard method for chemical analysis of antimony is the distillation-bromate method.
- In pig lead (ASTM E37-56), the method established for determining antimony is manganese coprecipitation.
- In ferroalloys (ASTM E31-73) a gravimetric method is described.
- In ASTM E396-72a, the Rhodamine B photometric method of chemical analysis is the standard.

Spot Tests

Spot tests are used by chemists to indicate the presence or absence of specific elements. Spot tests are not quantitative but they are frequently rated for the limits of concentration where they are effective. For spot tests (Feigl and Anger, 1972) (see also: Feigl and Chan, 1967a,b) a variety of analytical methods are offered for antimony.

- Metallic antimony forms a sublimate of volatile antimony^{III} chloride by means of pyrolysis with an excess of ammonium chloride.
- Elemental antimony can be detected to 100γ by a reduction-oxidation reaction with alkaline mercury cyanide solution. A strong alkaline solution of mercuric cyanide reacts with other antimony compounds, such as Sb_2O_3 or Sb^{III} salts, to form black or gray, finely divided metallic mercury. Salts of Sb^V must be reduced to Sb^{III} in acid medium before this reaction can be obtained. A limit of identification of 10_γ antimony has been obtained with potassium antimonyl tartrate using this procedure.
- Phosphomolybdic acid is used to determine Sb_2S_3.
- 9-methyl-2,3,7-trihydroxy-6-fluorone is used to determine antimony^{III}.
- Gallein gives a violet color in acid solution in the presence of antimony with a sensitivity of 0.005_γ.
- Rhodamine B, in tests similar to the ACGIH-AOAC method mentioned above, can be used to identify pentavalent antimony.

- Diaminotriphenylmethane dyes (Malachite Green and Brilliant Green) and triaminotriphenylmethane dyes (Crystal Violet) form colored dye complexes when used with antimony^{V} compounds in hydrochloric acid. These complexes are soluble in toluene.
- Antimony pentoxide and tetroxide remain following ignition of organic compounds containing antimony.
- Diphenylamine or N,N-diphenylbenzidine dissolved in sulfuric acid imparts a blue color in the presence of antimony to the limit of 5_{γ} antimony.
- Potassium antimony tartrate (tartar emetic of potassium antimonyl tartrate) may be detected using standard tests for trivalent antimony and tartrate. Limits of identification are 2.5_{γ} $K(SbO)C_4H_4O_6$ by forming antimony^{III} sulfide and 5.0_{γ} $K(SbO)C_4H_4O_6$ by silver oxide reduction.

By suitable selection of the concentrations of bromate, bromide and hydrogen ions, a technique to determine antimony III is described and compared to titrimetric and coulometric methods (Burgess and Ottaway, 1972).

A fluorometric method for the determination of submicrogram quantities of antimony using 3,4',7-trihydroxyflavone is described as being 25 to 50 times more sensitive than the Rhodamine B method (Filer, 1971).

A new flame analytical technique, molecular emission cavity analysis (MECA), is described for the determination of antimony (Belcher et al, 1974).

Forensic Tests

Traces of antimony of the order of 0.2 μg are deposited on the back of the hand from revolver ammunition each time the gun is fired, the source of the antimony being in the primer. A neutron activation procedure has been used (Ruch et al, 1964) to detect antimony after careful removal of the residues from the hand.

The apparatus for Reinsch's test was modified for rapid identification of antimony , arsenic, and mercury (Clarke et al, 1963).

Gutzeit Test

The Gutzeit method traces back about a century to the silver nitrate test for arsine. Mercuric chloride or bromide can be substituted for the silver nitrate reagent. The procedure involves the reduction of arsenic to arsine, using a few grains of zinc in a dilute sulfuric or hydrochloric acid solution. The gases, which evolve, pass through cotton-wool or over filter paper soaked in the reagent and result in a range of colored stains depending on the quantity of arsenic present.

The Gutzeit name was given to this test in 1879, when H. Gutzeit (Pharm. Zfg. 24, 263) devised a convenient apparatus for holding the filter paper in the gas stream. In this test stibine, present in amounts less than 0.1 mg, does not react. The presence of somewhat more stibine, however, causes the formation of a brown spot which is soluble in alcohol.

The so-called modified Gutzeit test, described in 1952 by V. Vasak and V. Sedivec ("Colorimetric Determination of Arsenic", Chem. Listy 46, 341) is similar to the ASTM method (ASTM D372-74) for testing arsenic in water. Both methods use a solution containing silver diethyldithiocarbamate to obtain a red compound with a maximum absorbance with a wavelength of 510 nm for antimony, when measured spectrophotometrically.

a. Analysis in Air

In the "tentative" method of analysis for antimony content of the atmosphere (E.C. Tabor, Chairman, Subcommittee 7, 1970) (Also in: Methods of Air Sampling and Analysis) the collected antimony must be oxidized to the pentavalent state before analysis with Rhodamine B is possible. The sensitivity of the antimony determination is 1.0 μg. In seven tests made, recovery of antimony ranged from 95-102.5% of the 2-10 μg sample.

The Rhodamine B method is also recommended by the Analytical Chemistry Committee of the American Industrial Hygiene Association and published by the Intersociety Committee Methods for Ambient Air Sampling and Analysis. The determination by this method is sensitive to 1 μg Sb in 10 ml solvent, or 0.05 $\mu g/m^3$ in a 20 m^3 air sample

Electroreducible cations of antimony can be determined by polarographic methods of samples collected from air (Dubois and Monkman, 1964). When there is little organic matter present and the sample is prepared carefully, polarography is a precise technique with sensitivity in the range that is adequate for industrial health purposes. The authors compare the method to dithizone-antimony analyses.

b. Analysis in Water

Atomic absorption is recommended by the Methods Development and Quality Assurance Research Laboratory (MDQARL) in the Methods for Chemical Analysis of Water and Wastes under standard conditions. The precision and accuracy reported by the MDQARL for a mixed industrial-domestic waste effluent at concentrations of 5.0 and 15 mg Sb/1. were ± 0.08 and ± 0.1 standard deviations respectively with recoveries of 96% and 97% respectively at these levels.

The test method for antimony listed in the Environmental Protection Agency Regulations on Test Procedures for the Analysis of Pollutants (40CFR 136; 38 FR 28758, October 16, 1973) is atomic absorption. A special notation refers to the MDQARL method.

Neutron activation analysis of antimony in six different marine fuel oils, before and after prolonged contact with seawater or fresh water, was performed by Guinn and Bellance 1969 (Skrinde, 1971).

There is no standard method described by the American Public Health Association (APHA), American Water Works Association (AWWA), and Water Pollution Control Federation (WPCF), for the examination of antimony in water and waste water in their joint publication.

c. Analysis in Biological Materials

A spectrochemical method was developed (Kinser et al, 1965) for detecting antimony in biological materials, following exposure to indium antimonide, the intermetallic compound, frequently used in electronic devices. At spectral line 2877.9 Å, concentrations as low as 0.1 μg of antimony can be detected with a coefficient of variations of 9.9%. Gamma ray spectrophotometry can be used to determine antimony deposits in the lung with a lithium drifted detector (Bloch, 1970).

Measurement of the quantity of antimony present is made by fractional transmission of X-rays at two different energies through the lung, one slightly less and the other slightly greater than the K shell binding energy of antimony.

A method using neutron activation analysis was developed (Mansour et al, 1967) to quantify the levels of antimony in blood and urine of patients treated with anti-bilharzial antimony drugs. This method was sensitive to 0.002 mg for antimony in biological fluids.

Thermal neutron activation analysis was combined with a chemical separation (Howie et al, 1965) to estimate antimony in small samples of biological material with a sensitivity of 10 μg to within 5%.

Hair and nails are a readily available source of biological sampling though the results require that many variables be considered (Hopps, 1974). For determining antimony III in human hair, as well as in several standard alloys, a method using forced-flow liquid chromatography and electrocatalyzed oxidation of Sb (III) in a platinum coulometic detector is described (Taylor and Johnson, 1974). Analysis of hair samples from four subjects, engaged in research involving antimony, showed correlation relative to exposure.

A method for determining antimony in blood and liver tissue (Loh and Cie, 1964) is described using Brilliant Green in a colorimetric analysis. The method requires several steps, but no interference from ion is reported.

2. Field Methods

A method for direct determination of antimony in sea water by anodic stripping voltammetry at the mercury-coated graphite electrode was developed especially for use on board ship (Gilbert and Hume, 1973). Typical results for the determination of the antimony content of sea water range from 0.18 to 0.48 μg Kg^{-1}, consistent with the values found by others using neutron activation analysis of freeze-dried samples.

For monitoring purposes, the Canada Centre for Inland Waters developed a method (Goulden and Brooksbank, 1974) for automated atomic absorption determination of antimony in natural waters. When a tube furnace is used as a covalent hydride decomposition device instead of a conventional hydrogen argon-entrained air flame, sensitivity is improved considerably.

Forty samples can be analyzed in an hour with a limit of detection of 0.5 μg/l. at a wavelength of 217.6 nm.

In a discussion concerning automation of monitoring equipment for marine pollution studies (Gafford, 1972), a method for automated analysis of heavy metals and trace elements is suggested. When using atomic absorption to analyze for antimony, the detectability limit desired is 0.005 ppm. A fluctional concentration limit (FCL) of 2.2. ppm is recommended with 15.4 as the practical lower limit of range for analysis (FCL X 7).

However, Halstead (1972) reports a virtual absence of environmental monitoring operations for toxic heavy metals as marine pollutants.

Analytical methods for determination of airborne particulate antimony are currently being developed at Arthur D. Little, Inc. (ADL) in conjunction with Standards Completion Program, Analytical Methods Validation for NIOSH. Methods are also being developed for stibine in air samples collection medium and analytical methods are currently being tested.

E. Monitoring

Antimony derived from the weathering of rock, was found in concentrations ranging from <0.0020 to 0.1 mg/l in selected drinking supplies in 39 U.S. states, listed on pp. 20-1 of EPA's "Water Quality Criteria Data Book, Vol. 2, Inorganic Chemical Pollution of Freshwater," Water Pollution Control Research Series, 18010 DPV 07, 71, July 1971. On p.13 of this publication, it is stated that antimony, if present in freshwater, would exist as the stibous form, Sb^{+++}, the stibic ion, Sb^{+++++}; or as triethylantimony, $Sb(C_2H_5)_3$.

Average values of antimony in the oceans have been estimated at 0.33 mg/l or 0.2 ppb (by weight). Antimony concentration in soil has been estimated at 4 ppm, while the concentration in the earth's crust is 0.2 ppb (by weight).

Portmann (1972), who gives the normal concentration of antimony in sea water as 4 μg/l, believes that the current exploitation of seabed resources will be expanded. In discussing the potential hazards from the extraction of minerals from the seabed, he comments that, although it appears unlikely that large concentrations of the element, antimony, would occur in sea water close to extraction operations, direct ingestion by marine animals cannot be ruled out. Because concentration factors of more than 300 have been reported for some marine animals, and as little as 97 mg have been reported lethal to humans, Portmann advises close monitoring of antimony extraction operations.

Antimony has also been detected in British waters. In a study prompted by current concern about the effects on marine organisms from localized inputs of heavy metals in domestic and industrial wastes, Leatherland and Burton (1974) report on measurements, by neutron activation analysis, of antimony and several other metals in organisms and bottom muds near Southhampton, England. Antimony was found in concentrations of only 0.01 to 0.1 ppm and the values for fish muscle were at the lower end of the concentration range. Concentrations of 0.2 ppm were found in two ascidians (sea squirts), a species with outstanding capacity for concentrating such trace metals as vanadium. Average concentration of antimony in bottom muds from Southhampton Water were almost 1.0 ppm; a River Mersey sample contained over 2 ppm.

Large amounts of wastes from mining and smelting operations, discharged into the Coeur d'Alene river in northern Idaho for more than 80 years, contain high levels of heavy metals. A recent study disclosed the presence of high concentrations of antimony, cadmium, copper, lead, silver, and zinc in the sediments of the delta area of the river, and it is reported that the lake bottom within a 900 m radius from the mouth of the river is covered with polluted sediments. Analysis of the top layer of the dry sediments (mostly fine silt typical of mine tailings) collected at the mouth of the river showed that antimony was present in a concentration range of 270 to 900 ppm. Other metals present were cadmium (16 to 75 ppm), copper (90 to 150 ppm), lead (3000 to 6300 ppm), manganese (6200 to 12,500 ppm), silver (6 to 15 ppm), and zinc (3200 to 4700 ppm). No toxicity attributable to antimony was mentioned, but earlier studies showed the accumulation of fair-sized amounts of lead and zinc in the tissues of some waterfowl and assumed that zinc was the lethal constituent of the wastes that caused the death of certain river fish in 72 hours (Maxfield et al, 1974).

By means of nondestructive neutron activation analysis, concentrations of antimony in the Sound surface sediments and at depths in cores range from 0.3 to 1.0 ppm dry weight, compared to 3-15 ppm for arsenic. Within 8-15 km of the smelter, antimony concentrations are 2-3 times the background values. Within 1 km of the smelter, sediments containing slag from the smelter contain up to 10,000 ppm of arsenic and antimony. Both wind and water distribute these metals from the copper smelter, but since the tidal current patterns do not transport the slag in the direction of Vashon Island, near Quartermaster Harbor, where 400 ppm of arsenic and 100 ppm of antimony have been found in the soil, they are evidently carried on the wind and settle out in that area. Arsenic to antimony ratios in the smelter stack dust are approximately 10:1; in the slag, about 1:1. However, since the ratio in sediments of Quartermaster Harbor is between 5:1 and 10:1, the slag is evidently not the major source of arsenic and antimony in the area.

Moderate tidal currents prevent the slag and the atmospheric particles from accumulating in the bottom sediment between the smelter and Vashon Island. Tidal currents transport the slag or dust to other area. In "noncontaminated" muds of Puget Sound about 50% of the Sb and most of the arsenic seems to be bound to extractable iron and aluminum compounds. Extractions of two contaminated sediments near the smelter indicated that under 20% of the arsenic and antimony is bound to extractable iron and aluminum. Most of the antimony appears to be bound in a relatively chemically stable form (Crecelius, 1975).

In September 1974, a NASA (Cleveland) news release indicated the presence of trace amounts of antimony, cadmium and other materials in Cleveland air. The levels found were not considered high enough to endanger human health. However, under contract to NASA, biologists from Central State University, will conduct a study to determine the potential long-term inhalation effects on rats of trace elements and compounds found in Cleveland's air.

A number of reports indicate that antimony is one of 30 or more trace elements, which may be emitted during the coal burning process. A recent study by the EPA National Fuels Surveillance Network found antimony among the trace elements in gasoline and in commercial gasoline fuel additives. Antimony, however, was not listed among the metallic elements (Ce, As, V, Ni, and Cr) in gasoline that are considered to have suspected biological toxicity (Jungers et al, 1975).

In 1966, the National Air Sampling Network recorded minute amounts of antimony in the air of cities and nonurban areas. Four of the 58 cities and three of the 29 urban areas analyzed showed antimony to be present in ranges of 0.042-0.085 $\mu g/m^3$ for urban and 0.001-0.002 $\mu g/m^3$ for nonurban air.

Efforts to obtain comparable data for more recent years have been unsuccessful. The National Air Sampling Network originally mailed filters from their headquarters in Cincinnati, Ohio to the some 200 to 250 volunteer workers who exposed the samples for the prescribed time and mailed them back. For some years after its relocation to Durham, N.C. more than five years ago, no filters were analyzed. Since then, the air sampling activity has become decentralized into the ten EPA regions and, in many cases, into state ambient air monitoring networks. Although each region operates to some extent differently from the others, there is a general effort to cooperate with the work in Durham. Apparently, the 1974 and 1975 data analysis will be worked on in order to be current. There will then be an attempt to analyze samples received but not analyzed during the years of relocation and reorganization. It appears that although some regional offices are running analysis of metals on filters, there are no antimony data being recorded by them at this time.

III. HEALTH AND ENVIRONMENTAL EFFECTS

A. Environmental Effects

1. Persistence and Environmental Accumulation

Antimony is found infrequently in air. Measurements of antimony in the atmosphere by the National Air Sampling Network in 1966 showed fair-sized concentrations in only six samples from 4 of 58 cities and in 3 of 29 nonurban areas in Arizona, Arkansas and New Mexico. The range of antimony concentrations reported in the four urban areas was 0.042-0.085 $\mu g/m^3$ and in the three nonurban areas was 0.001-0.002 $\mu g/m^3$ (Schroeder, 1970a; Woolrich, 1973). It is doubtful that such small amounts of antimony represent a hazard to human health.

Schroeder (1965, 1970) notes that the concentration of antimony as previously reported in the literature is 4 ppm in soil, 0.2 ppm (by weight) in the earth's crust and 0.2 ppb (by weight) in sea water.

Evidence for persistence of antimony in soil has been reported by Crecelius et al (1974) who showed that surface soil (upper 3 cm) samples obtained from sites 10 kilometers downwind of a Tacoma, Washington copper smelter contained 8-204 ppm Sb (dry weight). These levels were well above natural levels of antimony in soils from Puget Sound which were considered by the authors to be approximately 3-5 ppm.

Booz-Allen (1972) estimated that 500,000 tons of solid waste (mine waste and mill tailings) from the antimony industry had accumulated through 1968. This waste covered a 20-acre land area in Idaho.

In another instance (Maxfield et al, 1974), antimony was measured in a concentration range of 270 to 900 ppm in the fine silt collected at the mouth of northern Idaho's Coeur d'Alene river into which for over 80 years large amounts of wastes from mining and smelting operations have been discharged.

2. Bioaccumulation

Schroeder (1970a) has estimated that the daily human intake of antimony from air, utilizing the highest reported atmospheric concentration, is 1.7 µg maximum, while that from food and water is less than 100 µg, as reported by Howells in 1968. Murthy et al (1971) calculated a higher daily antimony consumption in a group of institutionalized children, ages 9-12, in 28 U.S. cities. The intake values determined in this study were 0.247-1.275 mg Sb/day. These levels were based on a weighted average antimony content in the diet, exclusive of drinking water, of 0.361 (0.209-0.500) mg/kg. The data indicated that the amount of antimony ingested varied significantly from one sampling period to another and from one institution to another.

Schroeder (1965, 1970) also reported the average value of antimony in the human body as less than 90 mg/70-kg man or less than 1.3 ppm by weight (using data from the ICRP Committee Report, 1960) and the total bodily content as 7.9 mg (according to a written communication from I.H. Tipton, 1970).

Several authors have analyzed human tissues for the quantitative determination of nonessential trace element content. In a paper by Brune et al (1963), the mean concentration of antimony in one sample of normal human whole blood was determined as 0.0044 (± 0.0021) µg/gm using a chemical separation technique with ion-exchangers combined with gamma-spectrometric analysis.

Radioactivation analyses of 45 samples of lung tissue obtained from adults of both sexes who resided within the Glasgow, Scotland area and who ranged in age from 40-70 years showed a mean concentration of 0.095 (± 0.105) ppm Sb wet weight. The maximum and minimum concentrations of antimony in these analyses were 0.452 and 0.007 ppm, respectively. The distribution was found to be log normal. Samples of 15 lung pairs showed that the mean concentration of antimony was more than twice as high at the apex of each lung as it was at the base, a finding which suggested that the source of the accumulated material was airborne dust (Molokhia and Smith, 1967). In 24 patients with pulmonary disease, Kennedy (1966) reported antimony concentrations ranging from <0.005 to 0.87 μg/gm lung tissue analyzed by neutron activation analysis.

The study by Molokhia and [illegible] (1967) also showed that in 15 tissue samples the visceral pleura contained a mean concentration of 0.037 ppm Sb (wet weight) or about the same amount of antimony as the base of the lung. In contrast, the antimony level in the trachea, pulmonary artery and vein, and tongue was very low (0.006-0.007 ppm) compared to that in lung tissue. The lymph glands analyzed contained 0.258-0.429 ppm Sb or about five to ten times more antimony than average lung tissue. The right hilar group was slightly higher in content than the left, and both were well above the paratracheal group.

Two investigators have analyzed involved and uninvolved lung tissue from patients with malignant pulmonary disease for antimony content by neutron activation analysis. The study by Kennedy (1966) indicated that the concentration of antimony was not increased in the involved and uninvolved lung of these patients in comparison to patients with nonmalignant pulmonary disease. In the other study (Molokhia and Smith, 1967), the concentration of antimony in tumor tissue was significantly less than that in the involved or uninvolved lung.

Human heart tissue obtained from autopsies of twenty victims of traumatic accidents showed a median concentration of antimony of 0.0015 μg/g wet tissue using neutron activation analysis. The range in concentration varied from 0.001 to 0.004 μg. The victims, who came from Stockholm and surroundings, ranged in age from 4 1/2 to 65 years. No significant differences in antimony concentration with age or sex were observed (Wester, 1965a).

Neutron activation analyses for antimony content were conducted by Hock et al (1975) on tissue samples from eight defined regions of six brains of humans aged from 5 hours to 74 years. The data were expressed in terms of element weight per unit dry tissue. The mean absolute antimony concentration in the cerebral cortex ranged from 0.25 to 17.07 x 10^{-7} g per g dried tissue. The mean absolute Sb concentration in the basal ganglia ranged from 1.58 to 18.71 x 10^{-7} g per gram dried tissue. The variation in concentration of nonessential elements including antimony in the different brain regions was found to be greater than the corresponding variations in the concentration of essential elements.

In a paper by Wester (1965b), four beef hearts, analyzed by neutron activation analysis, showed somewhat increased concentrations of antimony in the conductive tissue. Comparatively lower Sb concentrations were obtained in the ventricular septum than in the right atrium and conductive tissue. The concentrations of antimony, expressed in μg/gm wet tissue, ranged from 0.005 to 0.01 in the atrioventricular node, 0.003 to 0.01 in the bundle of His, 0.002 to 0.008 in the atrial tissue and 0.0007 to 0.002 in the ventricular tissue.

3. Biological Degradation

No information was found

4. Chemical Degradation

Chemical reactions of antimony compounds in the environment are discussed in Section II.C.7. The chemical degradation products of selected antimony compounds are given in Table I, pp. 6-8. For example, antimony trichloride reacts with water to form antimony trioxide and hydrochloric acid. The oxide may react with bases (in soil) to form metal antimonites, while the hydrochloric acid will form chloride salts. Analogous products are formed from other antimony halides. Antimony pentasulfide reacts with water to form hydrogen sulfide that may escape as a toxic gas, or react with bases to form sulfide salts. Other degradation products shown in Table I are non-toxic and of little interest.

5. Environmental Transport

Crecelius *et al* (1974) have reported that HiVol air filter dust samples collected in Seattle 40 km downwind of a Tacoma, Washington copper smelter often have extremely high concentrations of Sb (>300 ppm).

B. Biological Effects

1. Toxicity and Clinical Studies in Man

a. Occupational Studies

Industrial exposure to dusts of antimony oxides has been linked with an increased incidence of pneumoconiosis, with and without associated pulmonary dysfunction, among smelter workers processing antimony ore in France (LeGall, 1969), in England (McCallum, 1963, 1967, 1970), in the U.S. (Cooper et al, 1968) and in Yugoslavia (Karajovic, 1958).

LeGall has reported six to seven cases of pneumoconiosis in 41 workers (15-17% incidence) which occurred during the 1960's in a French foundry manufacturing antimony and antimony oxide from imported ores containing 1-20% silica. The pneumoconioses were diagnosed by X-rays which revealed moderately dense reticulo-micronodular foci in all pulmonary fields. Clinical signs were minimal and included cough and prolonged expiration; pulmonary function studies revealed a deficit. Antimony was not detected in the urine of four workers studied.

Concentrations of antimony in samples collected at the roasting furnace and at the furnace in which the fine oxide was produced were 0.30 mg/m^3. After prolonged sampling at a roasting furnace without ventilation, antimony concentrations ranged from 3.4-14.7 mg/m^3. Particle counts conducted in the mixing and roasting areas and in an area of furnace charging showed innumerable particles $\leq$ 3 microns in size. Duration of exposure of the affected individuals varied from 6 to 40 years. The authors commented that at certain points the concentration of SO_2 was repeatedly excessive (10 times higher than the admissible level) and that the fume deposits consisted of 3% quartz. Antimony was considered the principal causal agent of the lung changes although the intervention of silica and SO_2 was not denied.

Radiographic lung changes resembling the simple pneumoconiosis of coal workers (Category 1-3) were observed in 9% of a group of process workers involved in the production of antimony oxide and pure metal from sulfide ore in the UK (McCallum, 1963, 1967). These changes generally appeared to be symptomless, although one worker with penumoconiosis also had a clinical picture of chronic bronchitis with respiratory obstruction.

Examination of spot samples of urine from three men with lung changes showed excretion of 425, 480 and 680 μg Sb/liter. One furnace worker with pneumoconiosis, who retired from his job at the normal age of 65, showed urinary antimony levels of 55 μg and 28 μg/liter 7 months and 4 years, respectively, after leaving work. There was no detectable diminution in the lung changes over this period.

Skin irritation affecting particularly the forearms,thighs and flexures, where there was presumably chafing from clothing, was reported in warm weather. The condition, which was termed "antimony spots", consisted of papules and pustules around sweat and sebaceous glands. These spots disappeared rapidly over a weekend or holiday, but reappeared on return to work.

The atmospheric concentration of antimony determined at 10 sampling points in the area of the furnaces ranged from 0.53 to 5.34 mg/m^3 on several days during the three-week sampling period. All but three sampling points showed concentrations greater than 2 mg/m^3. The highest concentration was measured in the vicinity of the tapping operation at the metal furnace where a concentration of 36.7 mg/m^3 was monitored. The tapping operation was said to last for a relatively short period.

A follow-up study was conducted in the same plant in 1965-1966, three years after the original investigation (McCallum, 1967). In this survey, a more comprehensive radiological investigation revealed 26 new cases of antimony pneumoconiosis out of a total of 274 workers examined. Another 18 workers with the same condition were already under clinical observation as outpatients. The total incidence of pneumoconiosis diagnosed in this survey was 18%. The author commented that material for histological examination had been scarce. However, micropathological examination of sections from the lungs of an antimony worker who died from pulmonary carcinoma revealed an accumulation of dust particles and dust-laden macrophages lying in alveolar septa and in perivascular tissue without fibrosis or an inflammatory reaction.

X-ray spectrophotometry determinations of lung antimony content were carried out on 113 men who had been employed for 6 months or more in the antimony process (McCallum et al, 1970). The values obtained ranged from nil to just over 11 mg Sb/cm^2 of lung area in one worker with a total of 35 years employment. There was a significant association between length of employment and the amount of lung antimony, particularly when only the first 20 years of employment was considered. Radiographic category of pneumoconiosis was also directly associated with the mean period of employment and the mean direct lung antimony measurement, although in each category there was a wide variation in lung antimony content. Foremen and baghouse workers had higher median antimony levels than furnace workers while laborers and other workers had the least lung antimony.

In the report by Cooper et al (1968) the results of a study of antimony intoxication in a U.S. antimony refining plant in which workers were exposed to both the dust from antimony ore and antimony trioxide were presented. Pulmonary function studies were performed on 14 workers who had been exposed to antimony trioxide for periods of 1-15 years out of a total exposure population of 28 workers. These studies included vital capacity, lung volumes, minute ventilation, tidal volume, mixing efficiency, maximum-mid expiratory flow rates (MMEFR) forced expiratory volume in 1 second (FEV$_1$), maximum breathing capacity (MBC) and diffusing capacity. Of those individuals with pulmonary function abnormalities, one had definite small opacities, one had very early changes, and two had negative chest roentgenograms. The remaining three subjects with either suspicious or definite roentgenographic abnormalities all had normal pulmonary studies. Electrocardiograms were also monitored in seven workers, three of whom had antimony pneumoconiosis. Six of these seven workers showed normal tracings while one worker showed slight bradycardia.

Spot samples of urine were analyzed for antimony content in two surveys of 17 workers conducted in 1962 and in surveys of 15 workers conducted in 1965-1966. The results of these surveys together with roentgen findings from 13 of 28 workers are presented in Tables XI and XII. Antimony excretion appeared variable and without correlation to the roentgen findings. The authors commented that only one urine sample contained a level of antimony greater than the quoted "safe" concentration of 1 mg/liter, suggested by H.B. Elkins in *The Chemistry of Industrial Toxicology*, 2nd Ed., John Wiley & Sons Inc., New York, 1959, p. 246.

TABLE XI

ANTIMONY IN URINE
(µg./1,000 ml.)

Patient	1962 I	1962 II	1965	1966	Roentgen Findings
1	695	800			*
2	450	617	128	323	Suspicious
3	15	39			*
4	365	448	78		Positive
5	570	400	167	70	Suspicious
6	71	107	290	415	Suspicious
7	425	545	139	74	Positive
8	12	21			*
9	8	12.7			*
10	9	9.5			Positive
11					Negative
12	36	149			*
13	590				Negative
14	–				*
15	1,020	347			*
16	37	36			*
17	23	12			*
18	875	272			*

* No roentgenographic examination.

TABLE XII

ANTIMONY IN URINE
(µg./1,000 ml.)

Patient	1965	1966 I	1966 II	Roentgen Findings
19	118	772	735	Negative
20	77			*
21	20			*
22	34	116		*
23	176	707	0 (?)	Negative
24	43			Suspicious
25	222			Negative
26		333		Suspicious
27			0	*
28			0	*

* No roentgenographic examination.

Source: Cooper *et al*, p. 499 (1968). Copyright © 1968, Charles C. Thomas, Publisher. Reprinted by permission of Charles C. Thomas, Publisher.

Atmospheric concentrations of antimony monitored in 1966 at 36 locations in the plant under different environmental conditions ranged from 0.081 to 75 mg/m^3. The bagging operations were associated with the highest ambient Sb levels. Concentrations of antimony monitored in the area of these operations were 138 mg/m^3.

Karajovic (1958) reported that the pneumoconioses observed among smelting plant workers involved in the production of pure antimony in Yugoslavia was different in character and slower in onset than the pneumoconiotic changes from the mixed dust silicosis observed in mine workers. The smelting plant workers were exposed to a mixed dust containing 35.72-89.66% antimony trioxide and 1.26-6.20% antimony pentoxide. Other contaminants in the dust consisted of 1.12-11.50% total silicon, including 0.6-6.7% free SiO_2, 1.70-4.02% Fe_2O_3 and 0.32-9.16% As_2O_3.

Out of a total population (160) at risk, 101 smelting plant workers underwent on-site clinical examination. These workers, who were considered poorly nourished, were employed at the most dangerous positions for periods of more than 3 years. In addition to being exposed to considerable quantities of dust, they were also required to perform heavy physical work at varying temperature. The majority complained of slightly difficult breathing, fatigue at work, myalgia, light cough and slight dyspeptic discomfort without pain or diarrhea. In the summer, these workers complained of itching of the skin attended by rash.

Sixty-two of these 101 workers underwent radiographical examination of the lungs. The results were as follows: no pathological findings in 31 cases, 17 cases of type 'x', 14 cases of Pn. simplex and no cases of Pn. progressiva. Emphysema with bronchitis was established in 22 cases, in 8 of which the worker was under 40. The pneumoconiotic changes in these workers were limited mainly to the middle and lower sections, and numerous, small (0.5-1.5 mm) sharply defined spotty shadows predominated giving the lung fields a gravelly appearance. Emphysema was localized in the upper sections and directly above the bases. In contrast, the X-ray picture of the lungs of miners affected with mixed silicosis showed larger (> 3 mm), poorly defined and sparsely disseminated spotty shadows which were especially numerous in the intraclavicular sections. Symptoms of emphysema were localized chiefly in the lower sections.

Fifty-one of the 62 workers examined showed catarrhal symptoms in the upper respiratory passages. There were 16 cases of devatio septi nasi, 4 cases of tuberculosis secondary fibrosa, 12 cases of conjunctivitis and 16 cases of antimony dermatosis. Thirteen of these 16 cases involved workers at the reverbatory furnaces. The dermatosis was manifested by vesicular varioliform effluorescences which underwent necrosis in the center and left behind slight scars with hyperpigmentation.

Pulmonary ventilation function studies (vital capacity, expiratory reserve volume, maximal ventilation volume determined by the Kennedy and Tiffeneau tests) showed signs of slight insufficiency in 4 of 7 workers with pneumoconiosis and associated emphysema and no insufficiency in the remaining workers. ECG findings were normal in these workers although 5 workers exhibited hypertension.

Twenty of the original 101 workers examined underwent more detailed clinical examination in the hospital. Serological reactions (WR, Kahn, Meinicke) were positive in two workers; the SE, hemogram (Hb, RBC, L, Rtc, BPE, Heinz), hepatogram and proteinogram were normal. Using the method of Gutzeit, no antimony was found in the blood or urine; however, three workers showed antimony in the amount of 25 ug/liter urine with the polarograph method.

Gravimetric analysis (with electroprecipitator) of the work positions exposed to dust yielded values of 16-248 mg/m^3, and konimetric analysis (with thermoprecipitator) showed the presence of 2150-12,800 dust particles/cm^3. The particle sizes were predominantly under 0.5 microns. The authors commented they were unable to determine whether the clinical findings represented silicoantimoniosis or an accumulation of dust resulting in pseudonodulation.

Bulmer and Johnston (1948) reported no toxic effects in two workers aged 51 and 60 years handling antimony trisulfide in a laboratory for a period of six weeks or one year. The compound, which was considered exceptionally pure and which contained 0.18% lead and <0.07% arsenic, was being crushed, ground and screened to a desired particle size. Respirators were apparently provided but the authors commented that they were not used.

No subjective complaints were registered by the workers handling antimony trisulfide. Hemoglobin determinations and differential counts of the two exposed workers did not vary materially from those of another 8 workers who had no appreciable antimony exposure. Chest films of both men did not suggest dust effects nor was antimony detectable by a modification of Bamford's method in two urine samples taken from both workers. The antimony content of urine samples from 6 other workers with no appreciable exposure was also negligible.

Atmospheric samples obtained between the furnace and screening machine, and at the delivery end of the screening machine contained 42.0 mg and 52.0 mg antimony trisulfide/m^3, respectively. These concentrations were said to be typical of the general exposure during processing.

The authors concluded that poor absorption of antimony trisulfide dust of the particle size (unspecified) encountered in this study accounted for the low toxicity observed.

In contrast, Brieger et al (1954) reported an increased incidence of cardiovascular changes and ulcer among employees in a plant of the abrasives industry where resinoid grinding wheels were manufactured from phenol formaldehyde resins and antimony trisulfide. The population at risk consisted of 125 workmen who were exposed for periods of 8 months to 2 years. During the exposure period, six employees died suddenly, in addition to two other employees who died of chronic heart disease. Four of the decedents were under 45 years of age. No autopsies were performed, but in all but one case heart disease was implicated.

Since an occupational factor was suspected, a clinical examination was conducted on 113 employees. Of all men examined, 14 had a blood pressure of over 150/90 mm while 24 had a blood pressure of under 110/70 mm. Thirty-seven of 75 workers examined showed changes in the electrocardiogram, mostly of the T wave, which were considered significant. An X-ray study was also conducted since a large number of employees complained about GI disturbances. The study revealed an incidence of ulcer in 7 of 111 workers examined (63/1000) compared to 59 known cases of ulcer in the total plant population of 3912 (15/1000). Urine samples collected at random and examined by a procedure adapted from Frederick's method contained 0.8-9.6 mg antimony/liter.

Air tests using Frederick's method revealed the presence of 0.58-5.55 mg antimony trisulfide/m^3 with the majority of findings over 3.0 mg/m^3.

After the use of antimony trisulfide was abandoned, no further deaths from heart disease or abnormal increase in cardiovascular disorders were observed for several years. However, ECG changes in 12 of 56 reexamined employees showed a tendency to persist.

Taylor (1966) described an episode of acute antimony intoxication in 7 men after exposure to fumes containing antimony trichloride. The source of antimony exposure was leaking pumps from an enclosed process in which antimony trichloride was circulated at a high temperature and pressure as a 98% solution in anhydrous hydrochloric acid.

All seven workers suffered upper respiratory tract irritation which was attributed to hydrochloric acid. In addition, five of the men developed gastrointestinal disturbances including abdominal pain and persistent anorexia. These symptoms were slightly delayed in onset and were attributed to the systemic action of antimony. Routine blood counts including hemoglobin estimations and white cell counts were normal in four workers. Chest radiographs of all seven workers were also normal.

Urinary antimony analyses revealed a concentration in excess of 1 mg/liter in 5 of 7 exposed men, one or two days after exposure. The highest urine antimony concentration observed was 5.1 mg/liter in one subject two days after exposure. Intermittent analyses conducted on subsequent days showed a rapid fall in urine antimony content suggesting that urinary excretion of this element is relatively quick.

Subsequent environmental measurements taken 3 feet downwind from the leaking pump suggested that these workers were briefly exposed to an atmosphere containing up to 146 mg HCl/m^3 and 73 mg Sb/m^3.

A higher than expected rate of cancer of the respiratory tract in uranium workers of the Colorado Plateau was attributed to radioactivity in the mines. To determine if mining exposures other than radioactivity may have contributed to the genesis of the cancer, the mortality rates of 1759 miners exposed to low radiation levels were examined. Two cohort groups were studied. The 908 men in Cohort 1 had completed 15 years of mine work before 1 January 1937, the 851 in Cohort 2 had completed 15 years from 1 January 1937 through 31 December 1948. Both cohorts showed excess mortality from lung and heart diseases. In Cohort 1, there was a threefold increase in cancer of the respiratory system. Cohort 2 showed a statistically significant excess of cancers of the digestive system. Each cohort experienced an excess of cancers involving all systems other than the digestive and respiratory systems.

No obvious explanation could be drawn, except that a carcinogen was probably admixed in the air of the mines. The ore from the mines contained silica plus (in order of diminishing quantities) sulfur, iron, copper, zinc, manganese, lead, arsenic, calcium, fluorine, antimony, and silver. Of these, only iron and arsenic were considered suspect occupational lung carcinogens. Nickel was present only as a trace element; chromium and asbestos were not detected (Wagoner et al, 1963)

b. Epidemiology

No epidemiological studies were found in the literature reviewed.

c. Metabolic Effects Studies

Abdallah and Saif (1962) studied the effect of dosage route and regimen on the excretion rate and tissue deposition of sodium antimony dimercaptosuccinate in a group of Egyptian patients. Five groups of patients with five males each were given single doses of ^{124}Sb-sodium antimony dimercaptosuccinate at levels of 75, 100 or 125 mg Sb intramuscularly, or 75 or 100 mg Sb intravenously. These doses corresponded to an average concentration of 1.4, 1.7, or 2.1 mg Sb/kg body weight. Concentrations of ^{124}Sb were monitored in whole blood and urine using a scintillation counter, and in the liver, heart and thyroid by surface body scanning with a scintillation probe.

The results of analyses for urinary excretion of antimony are presented in Table XIII.

TABLE XIII

Excretion of antimony in urine in first 24 hours after a single dose of sodium antimony dimercaptosuccinate

Dose and route		*Hours*					
		0–1	0–2	0–3	0–4	0–5	0–24
75 mg.	mg. Sb	3·75	7·75	10·5	12·25	13	16·75
i.m.	%	5	10·3	14	16·3	17·3	22·4
100 mg.	mg. Sb	4·25	15·75	18·5	19·25	20	24·25
i.m.	%	4·25	15·75	18·5	19·25	20	24·25
125 mg.	mg. Sb	9·25	20·50	25·25	27·75	29	35·75
i.m.	%	7·4	16·4	20·2	22·2	23·2	28·6
75 mg.	mg. Sb	15·75		23·25		24	29
i.v.	%	21		31		32	38·6
100 mg.	mg. Sb	23·5		31·75		33·5	36·25
i.v.	%	23·5		31·75		33·5	36·25

Source: Abdallah and Saif, p. 291 (1962). Copyright © 1962, Little, Brown and Company. Reprinted by permission of Little Brown and Company (Boston, Mass.), Ciba Foundation, Churchill Press (London) and Dr. A. Abdallah

The urinary excretion of antimony was rather rapid during the first few hours, whether the drug was administered intravenously or intramuscularly. The total amount of antimony excreted in 24 hours increased with larger doses administered by either route. However, with similar single doses, larger amounts were excreted during the first 24 hours when the drug was administered intravenously. Long-term follow-up showed that with single intramuscular injections of 75, 100 and 125 mg Sb half the administered dose was excreted in the urine within 26, 15 and 7 days, respectively. With intravenous administration of 75 and 125 mg Sb, the 50% excretion level was attained on the 5th and 4th day respectively. Successive doses of antimony given intramuscularly or intravenously once weekly resulted in a more rapid initial excretion rate with an increase in the individual repeated dose, but only to a certain limit, after which any further increase was accompanied by a phase of relatively delayed urinary excretion.

Individual doses of 50 mg Sb (average 1 mg/kg body weight) were also administered every other day, intravenously or intramuscularly, for a total of five doses. With intramuscular injection, the urinary excretion rate was rather steady and 50% of the total dose administered was excreted within 30 days from the start of the treatment. In contrast, there was an initial rapid rise in excretion rate with intravenous therapy during the period of administration of the doses which was followed by a relatively slower excretion rate. Fifty percent of the total dose could not be recovered in the urine in 34 days.

With single intramuscular doses, the initial maximum level of antimony in the blood was attained within one hour after injection. This level had no correlation with the size of the administered dose. Another peak also occurred 2, 3 or 4 hours later. The timing of this second peak was apparently related to the decrease in blood concentration after the initial peak which it directly followed. The larger the individual dose, the less apparent was the decrease in the initial concentration of antimony in the blood in the first 5 hours. During the follow-up period of 28-50 days, there were minor transient elevations in the Sb concentration in the blood which did not represent any basal or critical level. With intravenous injections, a maximum level was attained in the blood within fifteen minutes after injection. This level did not appear to be correlated with the size of the administered dose. The maximum level was followed by a steep, then a steady step-wise decrease; no secondary peaks were observed.

With repeated doses, blood antimony levels were estimated one hour after intravenous injection and two hours after intramuscular injections. There was usually a tendency for the antimony blood levels accompanying successive doses not to exceed or even approach the average peaks attained by the first dose. Any intermittent rise was usually concommitant with a change in gradient of the excretion rate. Whenever the excretion rate changed to a slower gradient, the blood level rose then subsequently decreased.

Organ retention of antimony was studied in one patient who received a single intravenous dose of 100 mg Sb and who was followed for a period of 23 days. The largest uptake of antimony occurred in the liver followed by the thyroid and then the heart. The highest Sb level was attained in the liver and in the heart on the second day, whereas the thyroid had the maximum concentration on the first day. Elimination of antimony from the liver occurred rapidly, and markedly decreased around the 11th day, when it became approximately equal to the highest concentrations attained in the heart or the thyroid. Elimination of antimony from the heart appeared to occur in shifts during the first week. Around the 10th day, concentration in the heart was higher than that in the thyroid. It then became lower until the end of the follow-up period. In contrast, the retention in the liver increased in amounts with repeated doses of 100 mg Sb administered in three equal doses intramuscularly over a period of nine days.

ECG changes in Egyptian adults, adolescents and children treated for schistosomiasis with antimony dimercaptosuccinate (TWSb) have been reported by Abdallah and Badran (1963) and Davis (1961). In the paper by Abdallah and Badran, the course of treatment consisted of five daily intramuscular injections of 6 mg TWSb/kg body weight (total dose 30 mg/kg or about 7.5 mg Sb/kg) administered to 25 adult patients with normal electrocardiogram prior to treatment. ECGs were monitored after the completion of the treatment course. In five patients,ECGs were also performed half an hour after the first injection and after the third and fourth injections.

In seven of 25 patients, there was a significant increase in heart rate of 40 beats/minute or more. Only one patient showed tachycardia above 100 beats/minute. There was also a significant decrease in heart rate in four patients, but none of these showed a rate slower than 60 beats/minute. No changes in rhythm occurred.

There was a diminution in amplitude of the P wave, ranging between 25-50% of the original amplitude, in twelve patients. In two patients there was a prolongation of the PR interval to less than 25% of the original interval. There was a significant decrease in PR interval in four patients.

A decrease in the amplitude of the QRS complex, ranging between 10 and 40% of the original amplitude, occurred in 10 patients. In one patient there was an increase in amplitude of the QRS complex of 33%. Prolongation of the QRS interval of 20-35% of the original duration was noted in four patients.

In three patients there was a slight depression of the ST segment. A variable degree of changes in T wave occurred in 24 patients. In three of these patients the change consisted only in diminution of amplitude. In 21 patients there was inversion of the T wave in two to twelve leads. The T wave inversion was most common in leads III and avf. Prolongation of the Q-T interval not exceeding 25% of the original interval occurred in 10 patients.

A review of electrocardiograms showed that the changes were not due to acute effect of TWSb as there was no change immediately or up to two hours after the first injection. The effect of the drug on the myocardium was cumulative since it started after the third dose and was more marked after the 4th and 5th doses. Electrocardiograms reverted back to normal within four to six weeks.

In the report by Davis (1961), 19 male African children or adolescents, aged 11-20 years, weighing 26-57 kg, and with *Schistosoma mansoni*, *S. haematobium* or double infections, were treated with antimony dimercaptosuccinate intravenously. The total dosage ranged from 1.0 gm in 5 days to 2.0 gm in three days. Electrocardiograms were monitored before treatment, daily during treatment [illegible]or the first two or three days after treatment.

All patients showed inverted T waves in one or more leads following treatment. Those fifteen patients who had iso-electric or inverted T waves before treatment showed the onset of frank inversion or an increase in the amplitude of inversion following treatment. Inversion commenced at different times during treatment, bore no relation to the amount of antimony administered but was of maximal amplitude on the last day of treatment or during the first three days after treatment. Inversion was maximal in extent and amplitude in the right unipolar precordial leads.

Follow-up ECGs showed no abnormality in one case at 18 days, persistent abnormality in 7 of 12 cases at 28 to 33 days and in 2 of 5 cases at 54 days after treatment. This abnormality consisted of either persistent inversion of the T wave in the right unipolar precordial leads or the failure to regain their amplitude before treatment.

Prolongation of the QT_c interval was noted in nine of nineteen series of recordings. This change appeared on the last day of treatment or during the first three days after treatment. It was transitory, lasting about 2 days before reverting to normal.

The authors commented that the T wave inversion noted before treatment occurs among Africans of all ages and is a common finding among children. The changes observed following treatment did not differ from those previously described with the use of other therapeutic trivalent antimonials. The time of occurrence of maximal ECG change and the fact that the changes were largely reversible over a period of weeks, which roughly paralleled the excretion rate of residual antimony, indirectly supported the concept that the temporary myocardial damage resulted from accumulation of toxic trivalent antimony.

Abdallah and Badran (1963) noted that the recommended course of treatment with antimony dimercaptosuccinate (TWSb) produced more market electrocardiographic changes than the course of treatment with potassium antimony tartrate (tartar emetic), another trivalent antimony compound. (Abdallah and Badran, 1961). In the latter study, 20 Egyptian males with schistosomiasis and aged 14 to 45 years were administered 1 grain per 30 kg body weight intravenously every other day for a total of 12 treatments. The ECG changes produced with both compounds are summarized in Table XIV.

It is apparent from the table that P wave changes and changes in PR interval were more frequent following treatment with TWSb. While there was no change in duration of the QRS complex with tartar emetic, a prolongation occurred in 16% of the cases receiving TWSb. ST segment depression, not found with tartar emetic, occurred in 12% of the cases treated with TWSb. T wave changes occurred slightly more frequently with TWSb and the intensity of these changes were definitely greater with this compound. Inversion of the T wave in more than six leads occurred in 10% of the cases receiving tartar emetic and in 32% of those receiving TWSb.

TABLE XIV

Comparison of electrocardiographic changes in tartar emetic and TWSb

	Change	Tartar emetic %	TWSb %
Rate	Increase	45	44
	Decrease	15	36
P wave amp	Increase	—	—
	Decrease	40	48
PR interval	Increase	5	8
	Decrease	5	16
QRS amplitude	Increase	15	4
	Decrease	50	40
QRS duration	Increase	—	16
	Decrease	—	—
ST segment	Depression	—	12
T wave			
Diminished Amplitude		90 20	96
Inversion 1 to 2 leads		25	32
Inversion 3 to 6 leads			20
Inversion more than 6 leads		10	32
Q-T interval	prolongation	50	40

Source: Abdallah and Badran, p. 191 (1963). Copyright © 1963, The American Journal of Tropical Medicine and Hygiene. Reprinted by permission of The American Journal of Tropical Medicine and Hygiene

Abnormal electrocardiograms have also been reported in groups of patients with schistosomiasis following treatment with an intensive course of sodium antimony bis(pyrocatechol-2,4-disulfonate), another trivalent antimonial (O'Brien, 1959 and Zaki, 1955). In the paper by Zaki, 25 Egyptian patients received injections of the compound as a 6.3% aqueous solution twice daily for four days. Five ml (42.5 mg Sb) were administered per 60 kg body weight. The total dosage contained 340 mg antimony. ECG tracings were performed before and one hour after each injection.

After the third or fourth injection, electrocardiogram changes usually began to appear. TI and T4 become smaller in amplitude. These changes were progressive during the course of treatment and became more progressive in the few days immediately after the treatment course. In some cases changes in the ST segment in L4 were found. This finding was observed after the fifth injection. The changes in the ST segment were always in the form of elevation above the usual level.

In the report by O'Brien (1959), young, well-nourished West African soldiers with schistosomiasis received an intensive course of treatment with sodium antimony bis(pyrocatechol-2,4-disulfonate). A total of 95 ml of the drug was administered intravenously over a period of 20 days. After initial graded doses, five ml (42.5 mg Sb) were given daily. The total dose of antimony was 807.5 mg.

During the course of treatment, one man developed multiple Stokes-Adams attacks which were shown to be due to gross ventricular dysrhythmia by electrocardiogram. The patient recovered completely after administration of BAL (British Anti-Lewisite). Electrocardiograms recorded towards the end of the course of treatment showed abnormalities in all 20 cases. The abnormalities were remarkably constant and consisted of an elevation of the ST segment followed by a sharp inversion of the T wave in the right ventricular unipolar precordial leads. The T wave inversion sometimes extended over into the left ventricular leads. These changes were considered those of underlying heart muscle damage, apparently of a temporary nature, since the ECG recordings three months later were normal. Other toxic side effects of antimony administration such as anorexia, nausea, vomiting, substernal constriction and dyspnea were also recorded.

Sapire and Silverman (1970) have classified ECG changes noted following treatment with antimonials according to severity. These authors and Woodruff (1969) have commented that the relationship of dosage to ECG changes is unclear. Individual hypersensitivity to antimony and the type of antimonial used are considered more important factors than the total dose of antimony administered. In fact, the severest ECG changes have been found to occur with the smallest doses.

No reports were available on ECG changes occurring after administration of pentavalent antimonials. Davis (1961) has noted that electrocardiograph changes following treatment with pentavalent antimony compounds are much less severe than with trivalent compounds, since the latter are eliminated only slowly by the kidney. Pentavalent antimonials, in contrast, are metabolized by the liver and are excreted more rapidly (Sapire and Silverman, 1970)

d. Poisoning Incidents and Case Histories

Sapire and Silverman (1970) have reported a case of severe myocardial involvement in a 10-year old Bantu who was being treated for a urinary schistosomiasis. This patient was inadvertently given an overdose of sodium antimonyl gluconate in a dose of 300 mg daily for six days. After completion of the course of treatment, the patient suddenly began to convulse and severe vomiting ensued. During the convulsions, the heart rate was found to be rapid and irregular and the pulse became feeble and irregular. The ECG showed multiple ventricular extrasystoles with runs of paroxysmal ventricular tachycardia. The diagnosis of acute antimony poisoning with cardiotoxicity was made, the convulsions being due to Stokes-Adams attacks associated with the paroxysmal ventricular tachycardia. After initiation of chemotherapy, the ECG findings persisted for 48 hours although to a markedly reduced degree, whereafter the patient reverted to sinus rhythm. Subsequently his condition improved rapidly. The authors noted that the principal effects appeared in the ST segment and in the T wave with only occasional changes in the QRS axis being noted.

Woodruff (1969) has reported the autopsy results of a Nigerian patient who died following treatment with a standard course of sodium antimony tartrate. The total dosage of drug was 1.38 gm. Autopsy findings revealed an infarct of the posterior portion of the interventricular septum, with widely patent healthy coronary arteries. The infarct was interpreted as a result of transient coronary artery spasm, a pathology which was in accord with the observed ECG changes in other patients.

Cordasco and Stone (1973) have reported a case of acute antimony intoxication in a 39-year old man exposed to an undisclosed amount of antimony pentachloride following a gas leak from a reactor. The man suffered second and third degree burns over most of his body. Twenty-four hours after admission he became acutely ill with respiratory distress. Examination of the chest revealed marked moist rales in both basal and mid-lung fields. His condition gradually worsened, with the development of pulmonary edema and persistent progressive respiratory distress. At approximately six weeks postexposure, respiratory acidosis ensued. Subsequently the patient improved following long-term intensive respiratory care and repeated subglottic and tracheal dilatation.

2. Effects on Mammals

a. Absorption, Excretion and Tissue Distribution Studies

(1) Absorption and Excretion

Waitz *et al* (1965) have studied the excretion of a trivalent antimonial, tartar emetic, in mice and rats. Fourteen groups of 10 female Spartan or Carworth CF-1 mice were given a single oral dose of ^{124}Sb-labeled tartar emetic at a level of 8, 16 or 32 mg Sb/kg. Antimony levels in the urine and feces were determined at 1, 5 and 25 hours after treatment by scintillation counting. The results, presented in Table XV show that the concentrations in the feces were always greater than in the urine. This finding probably indicates incomplete absorption from the GI tract although biliary excretion was not ruled out.

TABLE XV

ANTIMONY LEVELS IN URINE AND FAECES OF MICE AFTER A SINGLE ORAL DOSE OF TARTAR EMETIC

Mice [a]	Dose (mg/kg)	Collection time (hours)	Urine		Faeces		Total excreted	
	Sb		µg Sb	% of dose	µg Sb	% of dose	µg Sb	% of dose
Normal	8.8	0-1	0.3	0.01	0.8	0.04	1.1	0.05
Normal	8.8	0-5	12.5	0.6	88.7	4.3	101.0	4.9
Normal	8.8	0-25	169.0	7.9	940.0	43.7	1 109.0	51.6
Infected	8.8	0-5	0.5	0.05	35.5	3.3	36.0	3.4
Normal	17	0-1	1.5	0.04	4.2	0.1	5.7	0.1
Normal	17	0-5	56.9	1.4	197.0	4	254.0	6.1
Normal	17	0-25	216.0	5.3	1 804.0	44.0	2 020.0	49.3
Normal	37.7	0-1	0.1	0	10.7	0.1	10.8	0.1
Normal	37.7	0-5	38.7	0.4	95.7	1.0	134.0	1.4
Normal	37.7	0-25	429.0	4.6	1 827.0	19.7	2 256.0	24.3

[a] 10 mice per group.

Source: Waitz *et al*, p. 538 (1965). Copyright © 1965, World Health Organization. Reprinted by permission of the World Health Organization, Dr. J. Allan Waitz and Dr. R.E. Ober

Wan-chu et al (1959) studied the gastrointestinal absorption and biliary excretion of antimonyl quinine hydrochloride, a trivalent antimonial, after oral administration in rats. Following treatment, antimony disappeared very slowly from the GI tract with about 90% of the administered Sb remaining after 8 hours. When the tract was ligated between the duodenum and pylorus prior to treatment, large amounts of antimony could be recovered from the intestinal tract within a few hours. The bile also contained antimony a few minutes after treatment. Sb excretion occurred mainly in the feces with less than 1% present in the urine.

In contrast, a large proportion of a dose of ^{124}Sb labeled tartar emetic was excreted in the urine of rats following intravenous administration. Six pairs of male Holtzman strain rats weighing approximately 200 gm were injected with 2.2 mg Sb (approximately 11 mg Sb/kg). Urinalysis for antimony content at 0.5, 2, 4, 8, 24 and 72 hours after treatment revealed a rapid urinary excretion with 73% of the dose being eliminated within the first 24 hours. Fecal excretion was 5.9% in 72 hours with less than 1% excreted in any of the time periods between 0 and 24 hours (Waitz et al, 1965).

Felicetti et al (1974a) have indicated a low gastrointestinal absorption of trivalent antimony in studies in hamsters. The radioactive labeled antimony used in this study was generated from a starting solution of ^{124}Sb-tartrate complex. Following oral administration to four hamsters, antimony was retained with a half-life of less than one day. The two animals that received the larger amounts of the trivalent compound retained 15% and 19% of their initial body burdens on day 4, of which 88% and 90%, respectively, were found in the GI tract. Low GI absorption was also indicated in experiments with the pentavalent compound.

The same authors studied the relative excretion of trivalent and pentavalent aersols of ^{124}Sb-tartrate following inhalation in hamsters. Both aerosols were generated from a starting solution of ^{124}Sb-tartrate complex and passed through a heating column at 100°C before introduction into the exposure chamber. The mean aerodynamic diameters of both aerosols were 1.6 microns with a geometric standard deviation of 1.7. Both exposures were "nose only". Urine and feces samples were collected for 32 days from seven hamsters in the pentavalent group and from seven animals for 15 days and five animals for 32 days in the trivalent group. Excreta were analyzed for ^{124}Sb content using deep-well liquid scintillation detectors.

No statistically significant differences in the excretion patterns were observed between the two aerosol groups. The inhaled trivalent and pentavalent antimony were excreted both in the urine and feces. In the early collections, more excreta activity was present in the feces than in the urine. The authors considered that upper respiratory tract clearance probably accounted for some fecal antimony in both cases.

Felicetti et al (1974b) have also studied the excretion of inhaled ^{124}Sb in the beagle dog as a function of temperature of aerosol formation. In this study three adult beagle dogs of approximately 15 months of age were exposed to aerosols generated from a ^{124}Sb-tartrate complex and passed through a heating column maintained at 100, 500, or 1000°C. The resulting particles had activity median aerodynamic diameters of 1.3 1.0 and 0.3 microns, with respective σ_gs of 1.6, 1.6 and 1.3. The dogs were anesthetized with pentobarbitol. The exposures were of a "head-only" type. Urine and feces collections were made on three dogs, one at each temperature of aerosol generation, for 32 days postexposure. Samples were analyzed in a deep-well liquid scintillation counter.

At least seven times as much ^{124}Sb was excreted in the urine than in the feces over the first 24 hours with the 100°C aerosol. In comparison, a urine to feces ratio of 0.4 over the same period was obtained for the 500°C aerosol. From day 2 through day 32, however, the relative compartmentalization between the urine and feces for all three aerosols was statistically the same. The mean urine to feces ratio for all the data combined was 0.81 over this period, indicating a relatively greater excretion in the feces.

The effect of pretreatment on the excretion pattern of subsequent doses of ^{124}Sb-labeled potassium antimony tartrate was investigated by Girgis et al (1965) in mice. Male, albino mice of a homogeneous stock, 8 weeks in age and weighing about 25 gm, were administered tartar emetic intraperitoneally. In the first series of experiments, the test groups received 35 mg/kg of unlabeled compound followed by 35 mg/kg of ^{124}Sb-labeled tartar emetic, whereas the control group received 35 mg/kg of ^{124}Sb-labeled drug. In the second series, the test group received 35 mg/kg of unlabeled tartar emetic followed by 70 mg/kg of ^{124}Sb-labeled drug, whereas the control group received only 70 mg/kg of ^{124}Sb-labeled drug. Urine and feces were collected during the five hour period after treatment with labeled compound and analyzed using a scintillation counter.

In the first series, excretion of ^{124}Sb over the five-hour interval was erratic, but fecal excretion in the test group greatly exceeded that of the control group and amounted to 17% of the dose. In contrast, urinary excretion of antimony was much higher in the test group in the second series and consisted of 37.2% of the administered dose. The fecal excretion was again found to be increased, although in some cases no feces were passed.

(2) Blood

The relationship between a single oral dose of ^{124}Sb-labeled tartar emetic and antimony blood levels in mice has been studied by Waitz et al (1965). The experimental procedure was described earlier (p. 101). The amount of antimony in the blood 1, 5 and 25 hours after treatment was linearly related to the dose administered, and decreased both linearly and quadratically with time. A significant interaction was found between dose and time. Blood antimony levels were not significantly different in normal and infected (with a Puerto Rican strain of _S. mansoni_) mice.

Data from blood samples in rats injected intravenously with 2.2 mg Sb (approximately 11 mg Sb/kg) as ^{124}Sb-labeled tartar emetic showed a rapid decline in antimony levels following treatment, a finding which supported the excretion data described previously (p. 102) (Waitz _et al_, 1965).

Bahner (1954) has studied the localization of antimony in the red cell fraction of rat blood. One male albino Wistar rat, weighing 386 gm, was administered 1.2 ml of dilute solution of labeled antimony catechol disulfonate complex (Fuadin) in 0.9% NaCl intravenously and sacrificed after 10 days. A sample of blood was defribinated and centrifuged and the hemoglobin isolated. The activity of the hemoglobin fraction was sufficient to account for all the activity in the defribinated blood. The author theorized that antimony was attached to the globin portion of the hemoglobin, since hemin isolated from pooled blood of ten rats, which had received intravenous injections of Fuadin 8 days previously, showed low radioactivity counts.

Waitz _et al_ (1965) also studied antimony blood levels in monkeys following a single oral or intravenous dose of ^{124}Sb-labeled tartar emetic. The oral dose was administered at a level of 8 mg Sb/kg to five Rhesus female monkeys weighing 2-3 kg. Blood samples were analyzed for antimony at intervals of 0.5-144 hours following treatment. Average peak blood levels of 0.175-0.180 μg Sb/ml were generally obtained 6-8 hours after dosing and individual values were as high as 0.25 μg/ml. Low but detectable blood levels of antimony were found 96-144 hours after dosing. The authors commented that these findings were in good agreement with the long period of urinary excretion of an intravenous dose in man.

Following intravenous injection of 1.28 mg Sb/kg to three monkeys, samples were taken at 0.25, 0.5, 1, 2, 4, 8 and 24 hours for blood antimony analyses. In all monkeys, blood antimony dropped rapidly from a high of 1.25-1.9 μg/ml at 0.25 hours, to 0.2-0.25 μg/ml at 8 hours, and to 0.1-0.2 μg/ml at 24 hours. The blood antimony curve in these studies was considered very similar to that described in man.

Blood antimony levels in mice and beagle dogs following inhalation of ^{124}Sb aerosols generated at various temperatures has been studied. In the first study (Thomas et al, 1973), three groups of 48 female Charles River CD-1 strain mice, 35 days of age, were exposed to aerosols generated from a ^{124}Sb-tartrate complex passed through a heating column maintained at 100, 500, or 1100°C. The respective mean aerodynamic diameters of the particles generated were 1.6, 0.7 and 0.3 with σ_gs of 1.9, 1.8 and 1.3, respectively. The exposures, which were about 10 minutes in duration, were of a "head-only" type. Whole blood samples at 2 days postexposure gave values of 0.43% of the adjusted body burden per ml for the 100° aerosol, 1.2% per ml at 500° and 1.0% per ml at 1100°. All values were essentially the same at 4 days postexposure.

Felicetti et al (1974b) exposed dogs in a similar manner (described previously, p. 103), and a differential concentration of ^{124}Sb in the RBC, compared to the plasma, was maintained for the entire 21 days of sampling. Ratios of RBC to plasma ^{124}Sb were generally similar for all aerosols, with the possible exception of a lower ratio at 100° during the first 24 hours. Beyond one day postexposure, the RBC concentrated ^{124}Sb by an average of 6.7 times that in the plasma throughout the sampling period.

The relative distribution of blood antimony between RBC and plasma following inhalation of the trivalent and pentavalent aerosols has been studied in hamsters (Felicetti et al, 1974a). The experimental procedure has been described previously. Blood samples were obtained at 0, 1, 2, 5 and 8 days postexposure from at least one animal per group by heart puncture. Distribution of blood antimony between RBC and plasma differed with the two valence states at early sacrifice times. Trivalent antimony concentrated in the RBCs at all sacrifice times with maximum concentrations of 6 to 10 times plasma levels on day 1 postexposure (approximately 24 hours). The pentavalent antimony group had plasma levels of about 3 times as great as the RBC levels at 2 hours postexposure, although antimony was concentrated in the RBCs 24 hrs postexposure. After the day 1 sacrifice, activity ratios from the two groups were similar. The activity half-times in RBCs were only a few days. The authors commented that establishment of a concentration gradient in the pentavalent group at 24 hours postexposure could have reflected some reduction to trivalent antimony in the blood and subsequent accumulation in the RBC.

The effect of pretreatment on blood antimony levels of subsequent doses of ^{124}Sb-labeled potassium antimony tartrate was investigated by Girgis et al (1965) in mice. The experimental procedure was described previously (p. 104). In the first series of experiments, the concentration of ^{124}Sb in the blood in animals which received a second dose of 35 mg/kg of labeled tarter emetic was similar to that of the control animals. When the second dosage was increased to 70 mg/kg, the blood antimony levels in the test group were found to be about 1/4 those in the control group. This difference was statistically significant.

(3) Whole-Body Retention And Tissue Distribution

Waitz et al (1965) measured concentrations of antimony in the liver of normal mice or mice infected with a Puerto Rican strain of S. mansoni following single or repeated oral doses of ^{124}Sb-labeled tartar emetic. The experimental procedure was described before (p. 101). Following a single oral dose in normal mice, the amount of antimony in the liver was found to increase linearly with the administered dose and quadratically with time. There was a significant interaction between time and dose which indicated that the relationship between liver antimony levels and dose was not the same at 1, 5 and 25 hours after treatment. Normal mice had significantly higher concentrations of antimony in the liver than infected mice. Antimony levels in the liver of 5 groups of infected mice given oral doses of 16 mg Sb/kg once daily for 2, 4, 6, 8 or 10 days were reasonably uniform from day to day and showed no persistent accumulation of the drug.

Molokhia and Smith (1969a) studied the tissue distribution of antimony in mice following a single intraperitoneal injection of tartar emetic or TWSb (identified by the authors as sodium antimony 2,3-mesodimercaptosuccinate; Astiban). BSVS male mice weighing 25-30 gm were infected with an Egyptian strain of S. mansoni. Fifteen mice were administered tartar emetic intraperitoneally in a dose of 5 mg/kg body weight. Fifteen other mice were given TWSb intraperitoneally in a dose of 7.5 mg/kg body weight. Both of these dosages contained the same amount of trivalent antimony. Two mice from each group were sacrificed at 0.5, 8 and 24 hours and 2, 4, 7 and 15 days following injection. Tissue samples were analyzed for antimony by neutron activation analysis.

The pattern of relative antimony uptake by the different mouse organs was shown equally by both groups treated with TWSb and tartar emetic. The highest antimony levels were recorded in the liver, GI tract, kidney and urinary bladder during the first 48 hours. The antimony content of the stomach and esophagus was significantly less than that of the duodenum and colon. The difference was probably due to antimony excreted via the bile into the duodenum. In the latter part of the experiment, the relative antimony uptake by the flat bones of the skull and by the teeth increased until it became higher than that of the other tissues. The brain, thyroid and male reproductive organs contained the lowest antimony concentrations throughout the experiment. The ventricular muscle of the heart, the tongue and the adductor muscles of the thigh all gave nearly identical values, and these were comparable to the antimony level in the blood. Skin levels were usually higher than those of muscle tissue probably due to binding of antimony with sulfhydryl groups in the skin.

Whole body retention patterns and tissue distribution of antimony were also studied in mice following inhalation of aerosols containing ^{124}Sb (Thomas et al, 1973). The experimental procedure has been described previously. Immediately following exposure and at intervals thereafter, the animals were whole-body counted in a liquid scintillation well detector. Serial sacrifices for tissue distribution were made at 0, 2, 4, 8, and 16 and 32 days postexposure.

The serial whole-body counts showed an early period of rapid clearance followed by a steady decrease in the rate of loss of ^{124}Sb. Retention patterns for the more insoluble 500 and 1100° aerosols were nearly similar, while the soluble 100° aerosol was cleared to a greater extent, particularly at early times. The difference in retention half-lifes (39 days at higher temperature vs. 29 days at 100°) was considered a function of the differences in tissue distribution.

Data for tissue distribution showed an approximate order of magnitude difference in lung tissue content between the 100° aerosol and those at 500 and 1100°. This difference was also reflected in the femur data and data from the carcass, a reflection of skeletal deposition. At the lower temperature aerosol, approximately 85% of the body burden at 52 days was deposited in the skeleton. The data from liver analyses did not reflect the pattern observed for skeleton.

The authors theorized that those particles at the lower temperature were more soluble, leaving the lung very soon after deposition and transporting in large degree to bone. The aerosol particles generated at the two highest temperatures tended to remain much longer in the lung, with less absolute accumulation in the skeleton, although a gradual buildup in the skeleton was indicated at all temperatures studied. The authors concluded that the overall retention, regardless of critical organ (lung or skeleton) would tend to place antimony in the class of compounds that have a retention time on the order of weeks.

Distribution of antimony in the liver and kidney following intravenous administration of ^{124}Sb-labeled tartar emetic has been studied in rats by Waitz et al (1965). Six pairs of male Holtzman strain rats weighing approximately 200 gm were injected with 2.2 mg Sb (approximately 11 mg Sb/kg). One pair was sacrificed at 0.5, 2, 4, 8, 24 and 72 hours after treatment and the liver and kidney analyzed with a scintillation counter. Kidney antimony levels were found to be higher than those in the liver, a finding which supported the rapid urinary excretion of antimony.

Mathews and Molinaro (1963) have also shown elevated Sb levels in kidneys of rats with a subcutaneous transplanted fibrosarcoma in a study which monitored the uptake of various radioactive substances used for brain tumor localization. Wistar-derived male and female rats, weighing 162 - 326 gm, were injected intravenously with $^{122}SbOCl$ or Na $^{122}SbO_2$. The radioactive uptake in the brain, tumor and other organs was monitored at 1 or 4 hours, respectively, after injection. The results showed that the distribution of ^{122}Sb was mainly extracellular since the blood concentration was usually higher than the concentration in organs and organ concentration was proportional to blood concentration. Exceptions were the kidneys, bone and spleen. With these organs the concentration at some time was greater than in the blood, and so presumably ^{122}Sb was taken up by cells or was actively concentrated in these particular organs. The kidneys of animals treated with $^{122}SbOCl$ contained 3.90% of the dose/gm while the kidneys of animals treated with Na $^{122}SbO_2$ contained 1.31% of the dose/gm.

In the study by Felicetti et al (1974b), the retention patterns for beagle dogs exposed to ^{124}Sb generated at various temperatures (described before p. 103) showed initial clearance of the 100° aerosol to be the most rapid, involving over 80% of the initially deposited material. The average long-term biological half-lives were 100, 36 and 45 days for the 100, 500 and 1000°C aerosols, respectively.

One dog from each group was sacrificed at 32, 64 and 128 days postexposure for tissue distribution studies. ^{124}Sb concentrations were highest in the lung, thyroid, liver and pelt. At 100°C the thyroid concentrated ^{124}Sb to a greater extent than any other tissue. With the 500 and 1000°C aerosols, the lungs contained the highest concentration of ^{124}Sb followed by the thyroid. The relatively larger contribution of the lung to the whole-body retention with the higher temperatures and lower particle sizes was considered indicative of the low upper respiratory tract deposition of the smaller particles. Clearance of the lung burden was also considerably slower with particles generated at 500 or 1000°. This finding was considered to reflect a lesser solubility of the higher temperature aerosols. The pelt concentrated ^{124}Sb to a rather larger extent and, because of its size, contained a considerable proportion of the body burden at all sacrifice times for all three aerosols. Skeleton and liver also concentrated ^{124}Sb.

Felicetti *et al* (1974) studied the comparative whole-body retention and tissue distribution of trivalent and pentavalent aerosols in hamsters. The experimental procedure has been described previously. Whole-body clearance of both aerosols following inhalation occurred in two phases. The initial clearance was very rapid. The average body burdens at day 1 postexposure for the trivalent and pentavalent groups were 65% and 60%, respectively, of the activity found in the day 0 sacrifice animals. Early rapid clearance was followed by a slower clearance phase during which the remaining antimony was eliminated with a biological half-life of about 16 days.

By two hours postexposure, less than 1% of the body burden of either aerosol remained in the lungs. Tissue deposition patterns for the two aerosol groups were similar with the greatest concentrations of antimony occurring in the liver, skeleton and pelt. There was, however, significantly greater deposition of activity in the liver with the trivalent than with the pentavalent antimony. This difference was most pronounced on day 5. By day 32 postexposure, the livers of the trivalent and pentavalent groups contained 11 ± 4% and 5.6 ± 1.8%, respectively, of the total tissue. Activity per gram in the femur was significantly greater than the muscle. Skeletal values were higher in the pentavalent than in the trivalent group, but differences were not significant until day 32 when the femur contained 3.4 ± 1.4% of the total tissue activity/gm of tissue in the trivalent group, and 6.0 ± 1.8%/gm of tissue in the pentavalent group. For both aerosol groups, a large percentage of the body burden was contained within the pelt at all sacrifice times from day 1 through day 32.

Valence state affected the relative distribution of antimony. In the pentavalent group, mean activity concentrations in the femur exceeded those of the liver at all sacrifice times. However, in the trivalent group mean concentrations were greater in the liver than in the femur until after day 15, when they were higher in the femur. The authors considered that no extensive reduction of pentavalent to trivalent antimony occurred, since a greater concentration of antimony in the liver was observed in the group that inhaled trivalent antimony than in the pentavalent group.

The effect of pretreatment of the subsequent tissue distribution of ^{124}Sb-labeled tartar emetic in mice was investigated in a study by Girgis et al (1965). The experimental procedure has been previously described. At the 35 mg/kg level, the concentration in the liver was similar to that in the control group which had no antimony pretreatment. ^{124}Sb concentrations in the heart, spleen and kidney, however, were found to be significantly lower in the test group than in the control. When the second dosage was increased to 70 mg/kg, the tissue levels in the test group were about 1/4 those in the control group except for the liver, in which the concentration was approximately 1/2 that of the control group.

b. Pharmacology

Antimony is an effective agent against such tropical infestations as schistosomiasis and leishmaniasis with a rapidity of action indicative of action beyond worm destruction alone. A study in young female CF mice infected with schistosome eggs demonstrated that treatment with potassium antimony tartrate (tartar emetic) i.p. at 20 mg/kg/day caused marked suppression of the granulomatous response to the eggs. Treatment also suppressed delayed foot pad swelling in response to soluble egg antigens injected into mice presensitized with schistosome eggs (Mahmoud & Warren, 1974).

A study of the specific effects on cardiac function in the intact dog demonstrated a progressive decrease in contractile force and a fall in blood pressure with death occurring within 120 minutes following an intravenous dose of 30 mg/kg body weight of potassium antimony tartrate (Bromberger-Barnea and Stephens, 1965).

In another study in dogs (Cotton & Logan, 1966) 7.5 mg/kg/day of tartar emetic or 10 mg/kg/day sodium antimony dimercaptosuccinate (TWSb) were given intramuscularly daily until death. Neither drug had much effect on diastolic blood but TWSb increased systolic pressure after 4-5 days of treatment. Both drugs caused a marked progressive increase in heart rate. There were no significant ECG changes during the first day of treatment with either drug, but changes occurred on the second day with tartar emetic and the third day with TWSb. The most consistent changes were tenting of the T wave and depression of the ST segment. Death occurred after 3-5 doses of tartar emetic or 5-7 doses of TWSb. When geometrically increasing doses of the two drugs were given to anesthetized mongrel dogs every 15 minutes doses of 1 mg/kg and 2 mg/kg had little effect on heart rate, blood pressure, right ventricular contractile force or hematocrit. With increasing doses blood pressure decreased progressively. Ventricular contractile force decreased immediately after each injection, but then slowly recovered. Deaths occurred after cumulative doses of 31-63 mg/kg with tartar emetic and somewhat higher doses with TWSb. Geometrically increasing doses of the two drugs given at 32 minute intervals, progressively and markedly reduced pressor responses to 1 µg/kg norepinephrine and progressively diminished reflex cardiac slowing, but did not affect the cardiac contractile responses. Pressor responses to 100 µg/kg tetramethylammonium bromide (TMA) were reduced progressively by the antimonials, but the increase in contractile force and cardiac slowing were relatively unaffected. Vasodilator effects of isoproterenol, acetyl choline and histamine were reduced as was the change in heart rate produced by isoproterenol, but cardiac contractile responses to isoproterenol were not affected.

In anesthetized dogs, electrical stimulation of the right vagus nerve was carried out before and after administration of geometrically increasing doses of tartar emetic and TWSb. There was no evidence of any significant change in the sensitivity of the sinoatrial node to vagal stimulation.

Heart rate was decreased somewhat only with the 64 mg/kg dose of tartar emetic.

The effects of antimonials on the tracheobronchial tree were studied in dogs by El-Hawey et al (1971). The trachea of each chloralose-anesthetized dog was surgically exposed and a Y-shaped cannula was fixed in the lumen. The animals were artificially respirated while the intercostal muscles and diaphragm were paralyzed with intramuscularly administered flaxedil. The antimonials, antimony dimercaptosuccinate (astiban) and potassium antimony tartrate (tartar emetic) were given intravenously at doses of 0.05-0.1 gm/kg and 0.4-0.6 ml 6% solution/kg, respectively, to 10 dogs each. Astiban did not produce bronchodilation in any of the dogs, but tartar emetic caused bronchospasm in 2 of the 10 dogs. From these results the authors conclude different modes of actions for the drugs: astiban with a direct relaxant effect; tartar emetic with a central vagal stimulatory effect.

c. Acute Toxicity

Parenteral - Studies of acute parenteral toxicity of trivalent antimony compounds in mice (Ercoli, 1968, 1971) indicate that toxicity is dependent on the chemical form of the compound administered. The LD_{50} values of several antimony compounds administered subcutaneously were in mg/kg (mg Sb/kg): potassium antimony tartrate 55 (20), sodium antimony tartrate 48 (19), sodium antimony tartrate-dimethyl cysteine chelate NAP 390 (57), sodium antimony tartrate - dimethyl cysteine chelate TP15 350 (51), sodium antimony tartrate-dimethyl cysteine chelate TP2 600 (73), stibocaptate 2,000 (500) and stibophen 670 (110). Administration by other parenteral routes did not greatly alter the toxicity: potassium antimony tartrate had an LD_{50} in mice of 49 mg/kg (18 mg Sb/kg) when administered intraperitoneally (Girgis et al, 1965) and 65 mg/kg (24 mg Sb/kg) when given intravenously (Ercoli, 1968), sodium antimony tartrate administered intraperitoneally 60 mg/kg (24 mg Sb/kg), the chelate NAP given intramuscularly 325 mg/kg (48 mg Sb/kg) and intraperitoneally 450 mg/kg (66 mg Sb/kg). Intravenously, the chelates TP15 and TP2 had LD_{50}'s of 450 mg/kg (65 mg Sb/kg) and 600 mg/kg (73 mg Sb/kg) Ercoli, 1968). With all compounds the toxic signs preceding death were weakness and difficulty of ambulation and respiration.

When rats and mice were dehydrated by deprivation of water for 48 hours or by replacement of drinking water with 2% saline for two weeks, deaths occurred more rapidly following intravenous injection of 1.0-1.5 mg/100 gm body weight of potassium or sodium antimony tartrate and mortality was significantly greater than in non-dehydrated animals. Both dehydration and antimony produced a fall in blood pressure until an hour before death. Blood transfusion preceding antimony injection did not counteract the effects of dehydration (Baetjer, 1969, 1973).

Oral - The oral toxicity of antimony trioxide was studied in rats. A single dose of 16 gm Sb_2O_3/kg produced no apparent ill effects during a 30-day observation period. Rate of growth and food consumption were similar to controls (Gross *et al* 1955a).

Inhalation - A single exposure of dogs and cats to 1 hour at 40-45 ppm stibine, antimony hydride, was reported to be "dangerous" to most of the animals. (Webster, 1946). Death generally occurred in less than 24 hours. Animals which succumbed showed pulmonary congestion and edema accompanied by marked hemoconcentration. Guinea pigs were more resistant with concentrations three to four times higher required to cause death within 24 hours (Webster, 1946). Changes in morphology of erythrocytes was observed rapidly with the appearance of spherical erythrocytes with tiny spicules extending symmetrically around the periphery. Within 10-20 hours hemoglobinuria occurred in most guinea pigs exposed to 65 ppm stibine for 1 hour. At sublethal doses, tolerance was apparently acquired rapidly. In animals which showed marked responses, fatty metamorphosis of the liver and splenomegaly appeared frequently.

In mice exposed in plastic chambers the LC_{50} of antimony pentafluoride was 0.27 mg/l at both high and low humidity (Chekunova & Minkina, 1970). At lethal concentrations, massive hemorrhages of the lungs and moderate degenerative changes of the myocardium were observed. In mice dying 5-10 days after exposure, the changes observed were degenerative dystrophic alterations of liver and kidneys, early signs of interstitial myocarditis, pulmonary congestion and hyperplasia and partial desquamation of the alveolar epithelium of the lung.

In a study by Gross et al (1969), pulmonary macrophage mobilization in rats and hamsters in response to different dust burdens was determined quantitatively in the absence or presence of mineral oil. Two groups of rats with 60 animals each received 1200 mg Sb_2O_3/m^3 by inhalation for a 12-hour exposure period or 30 mg of an aqueous suspension of Sb_2O_3 by intratracheal instillation. The average particle size of the dust was 1.1 microns. In addition, one half of both experimental groups received a small amount of mineral oil intratracheally (0.2-0.25 ml) in addition to the dust and the remainder received an equal volume of water intratracheally in addition to the dust. Hamsters received Sb_2O_3 in the same manner in concentrations of 900 mg/m^3 by inhalation or mg by intratracheal instillation. Lung macrophage determinations were conducted on two animals from each group at 0 hour, 1 day, 4 day, 1 week, 2 week and one month after treatment.

The percentages of macrophages recovered from both treated and control animals were not only variable, but unpredictable, particularly in animals that had been given a lung dust burden. Intratracheal injection of Sb_2O_3 dust into both hamsters and rats resulted in a significant increase in the mean macrophage count as determined by the saline washout method. Inhalation of Sb_2O_3 dust in rats and hamsters was also associated with significant increase in the mean macrophage count; the inhalation of Fe_2O_3 or SiO_2 dust did not result in a significant increase in either species. In rats, no significant increase in the mean macrophage count was observed when intratracheally injected Sb_2O_3 was associated with mineral oil. In hamsters, the simultaneous presence of mineral oil with Sb_2O_3 in the lungs was associated with considerably greater increases in the mean macrophage count than was found in this species with the dust alone. Although the increase in mean macrophage count was more sustained in the presence than in the absence of the mineral oil, there were fluctuations of some magnitude in the counts over the one-month observation period.

Eye Irritation - Antimony trioxide with particle size 1.3 ± 1.65 microns instilled into the right eye of male albino rabbits caused no irritation to conjunctive or cornea up to seven days after treatment (Gross et al, 1955a).

Cutaneous - An aqueous paste of 24 gm of antimony trioxide in methyl cellulose was applied to denuded skin of albino rabbits over two thirds of the torso, covered with an impervious membrane and allowed to remain in contact with the skin for 1 week. No local or systemic effects were noted (Gross et al, 1955a).

Dry antimony trioxide powder packed into incisions on the shaved backs of rabbits delayed healing to a degree expected with any nonspecific foreign body, but did not have any specific untoward effects (Gross et al, 1955a).

Intradermal injection (dosage specified only as "various concentrations" by the author) on the shaved back of rabbits of 6-8% water solutions of antimony-dimethyl cysteino tartrates caused only slight hyperemic reactions which disappeared within 24-48 hours (Ercoli, 1968). However, intradermal injection of potassium antimony tartrate was irritant at a concentration of 0.124% and at 0.5-1.0% produced intensive hyperemia followed within 3-4 days with a necrotic ulcer which healed in 4-6 weeks (Ercoli, 1968).

d. Subacute Toxicity

Parenteral - In a study of the cumulative toxicity of trivalent antimony (Ercoli, 1968) mice were treated subcutaneously for 10 days with 7.23 mg/kg of Sb as potassium antimony tartrate (tartar emetic) or 24.4 mg/kg of Sb as the dimethylcysteine-potassium antimony tartrate chelate TP2. Both groups survived treatment. All mice receiving tartar emetic were reported to have cutaneous lesions, but these lesions were not described. Mice receiving TP2 were reported to have had no signs of toxicity.

In dogs injected intragluteally for 7-9 successive days with 6% TP2 solutions corresponding to single doses of 1.45-2.4 mg Sb/kg (total dose of 10.2-14.5 mg Sb/kg), the drug was reported to be tolerated without significant side effects or change in appetite or behavior. Hematological examination revealed essentially normal results (Ercoli, 1968). One week after completion of the above course of treatments the dogs were given a second course of treatment which was continued until most of the dogs died. The total amount of Sb received, considered by the investigator to be the lethal dose, was 25 mg Sb/kg.

In a study of a pentavalent antimony drug, RL712, a stable non-ionic complex of antimony hydroxide and a partially depolymerized dextran glycoside, was given to female NMRL mice by intramuscular injection in the right or left hind leg alternatively three times a week for 17 weeks for cumulative doses of 28.2 or 80 mg Sb/mouse (Bou Casals, 1972). No effect on growth was observed and histological examination two weeks after the last injection of the higher level of Sb showed normal appearance of the injected areas, heart, spleen, liver and kidneys. Mice given the lower dose showed no signs of growth impairment or toxicity up to 26 weeks after the last injection.

Inhalation - In a study of the inhalation toxicity of particulate antimony trisulfide (particle size $\leq$ 2 microns) male Wistar rats were exposed to about 3.07 mg/m^3 for 6 weeks, 5 days/week, 7 hours/day (Brieger et al 1954). All animals survived the exposure; appearance and weight gain remained essentially normal throughout. However, definite and consistent changes in ECG were observed in all exposed animals, chiefly elevation of the RS-T segments of all leads, occasional low voltage of the QRS complexes and frequent flattened T waves mostly in Lead I. These changes were more pronounced on the last examination prior to termination of the experiment than on interim examinations. At autopsy the hearts of two rats were dilated due to flabbiness of the myocardium while the other hearts appeared normal. The lungs of all animals showed mild congestion and focal areas of hemorrhage. Histologically, all hearts showed slight to moderate hyperemia and most showed parenchymatous degenerative changes.

In rabbits exposed to about 5.6 mg/m^3 of antimony trisulfide for 6 weeks, 5 days/week, 7 hours/day (Brieger et al, 1954), no effects were observed on behaviour, survival, blood sugar and BUN levels, liver function tests, hematology or blood pressure. ECG's indicated slight to moderate myocardial damage with a tendency to progress. At autopsy there was marked dilation of the heart; the myocardium was very flabby and red-brown. Histologically there was swelling of the myocardial fibers and the presence of cytoplasmic granules.

Dogs exposed to about 5.32 mg/m^3 of antimony trisulfide for 7 weeks, 5 days/week, 7 hours/day (Brieger et al, 1954) showed no definite or consistent effects in clinical, biochemical, hematologic or electrocardiographic parameters. However, dogs exposed to a concentration of about 5.55 mg/m^3 for 10 weeks, 5 days/week, 7 hours/day showed some changes in ECG suggestive of myocardial injury. At autopsy there were no gross pathological findings. Histologically there was occasional swelling of myocardial fibers which possibly was associated with treatment.

Rabbits exposed to 27.8 mg/m^3 for 5 days survived, but many had ECG changes indicative of myocardial damage or coronary inadequacy. Slight to moderate parenchymatous changes of the myocardium were evident histologically. Slight degenenerative changes were also observed in liver and kidney and inflammatory changes in the lung (Brieger et al, 1954).

In another study of inhalation toxicity, albino Sprague Dawley rats about 84 days old were exposed to aerosolized antimony as antimony trioxide or powdered ore at a concentration of 1.7 gm/m^3 in chambers in which only the nose of the rats was in contact with the antimony. The animals received 1 to 6 exposures of 1 hour each, every 2 months for 66 to 311 days in the case of the trioxide and 66 to 366 days for the powdered ore. With the antimony ore, the lungs of some rats immediately after exposure showed generalized pulmonary congestion with mild edema but this response was predominantly transitory and nonlethal. Most rats survived. Except for the acute response pathology was similar in all animals exposed to either the trioxide or the ore. At 66 days, the pulmonary pathology consisted of a phagocytic response. Generally the dust-laden phagocytes were lying free in the alveolar spaces. At more prolonged intervals after exposures focalization of the dust into small deposits throughout the lung became increasingly prominent. Even at 311 days for trioxide and 366 days for the ore the phagocytic response persisted without any appreciable chronic pneumonitis. In the tracheobronchial lymph nodes there were scattered deposits of intracellular antimony with mild hyperplasia but no evidence of chronic inflammation (Cooper et al, 1968).

Exposure of rats to antimony pentafluoride gas for 2 hours/day for 3-1/2 months at a mean concentration of 0.015 ± 0.0014 mg/l SbF_5 (0.0085 ± 0.0008 mg Sb/l) caused no deaths or alterations in body weight or nervous system function as medicated by summation threshold index. Liver function was affected by the end of 3 months as indicated by decreased excretion of hippuric acid. Bradycardia occurred periodically during treatment and elevations of the ST and T waves of the ECG were noted. At autopsy an increased heart/body weight ratio was noted and histologically there were changes in the myocardial fibers. Histological changes were also observed in lung, liver, kidney and thyroid tissues. These changes were considered by the investigators to be related to both antimony and fluoride (Chekunova and Minkina, 1970).

e. Other Chronic Effects Studies

In a study in rats (Gross et al, 1955a) the animals were fed a synthetic basal diet to which 2% antimony trioxide had been added for about 34 weeks. These rats gained weight more slowly than pair-fed controls. At autopsy no gross or microscopic abnormalities in tissues or organs were observed.

Addition of antimony as potassium antimony tartrate to the drinking water of Long Evans strain rats at a concentration of 5 ppm for the lifetime of the animals (Schroeder et al, 1970) caused no consistent effect on growth or mature weights of either males or females but significantly reduced the longevity. Curiously, nonfasting serum glucose levels were lower than fasting levels in both sexes. Serum cholesterol levels were higher in males and lower in females than in controls. At autopsy heart weight was somewhat higher than controls in males, but this was not considered to be significant by the investigators. Antimony was found to accumulate in the tissues with age.

In a similar study in mice (Schroeder et al, 1968), 5 ppm antimony as the potassium tartrate in drinking water caused decreased medial life span and longevity and some suppression of weight in older animals in females only. There was no increase in hepatic fatty degeneration compared with controls. Increased levels of antimony were found in the tissues.

In a study of chronic inhalation toxicity of antimony (Gross et al, 1955b), rats and rabbits were exposed to air containing antimony trioxide (particle size about 0.6μ) at concentrations of 100-125 ppm and 85 ppm, respectively, for 10 hours/month for 14.5 months and 10 months, respectively. Beginning after 5 months exposure in rabbits and 9 months in rats the lungs showed mottling with chalk-white foci 1-2 mm in diameter. The cut surfaces were coarse-textured and dry. The mottling increased with longer exposure. Microscopically, inhaled Sb_2O_3 dust was early associated with swelling, proliferation and desquamation of alveolar macrophages. With longer exposure fatty degeneration of the macrophages became prominent. At the end of the experiment colorless needle-shaped crystals were observed in the air spaces, possibly cholesterol, and fibrosis was evident. Antimony accumulated in the lung tissues.

Implantation of irregularly shaped pellets (3-5 mm diameter) of pure antimony metal into the superficial precentral motor cortex of the cerebral hemispheres (right hemisphere or both) of three monkeys (*Macaca mulatta*) caused spontaneous clinical seizures in all three monkeys and clinical seizures were also observed following challenge doses of pentamethylene tetrazole (24 mg/kg) given intramuscularly and picrotoxin (0.35 mg/kg) given intravenously. EEG showed persistent slowing in all three monkeys. Two of the monkeys died after 2 months; the third survived for a year. In all three there was a necrotizing foreign body reaction in the brain with a grey-white necrotic soft spot of 5 mm diameter surrounding the metal fragment (Chusid and Kopeloff, 1962).

f. Tolerance - Repeated Doses

In a study in 8-week-old mice given potassium antimony tartrate by intraperitoneal injection (Girgis et al, 1965), mice which survived single high doses were markedly resistant to subsequent high doses. Maximum tolerance was developed within one day after the first injection and was reduced considerably after a week. However, some tolerance remained for a long period. For example, a second dose of 56 mg/kg given 60 days after a dose of 35 mg/kg caused death in 1/19 mice (single dose LD_{50} = 49 mg/kg). Tolerance was found to be reactivated by administration of another large dose.

g. Teratogenicity

In a study of the effects of a pentavalent antimony drug RL712 (Bou Casals, 1972) Wistar rats were given 125 or 250 mg Sb/kg body weight as an RL712 solution intramuscularly on five occasions between Day 8 and Day 14 of gestation. No effects were noted on numbers of fetuses, resorptions or implantations and no abnormalities were observed in the fetuses. Analysis failed to detect any antimony in the fetal tissues.

In rats exposed to antimony trioxide dust at a concentration of 250 mg/m^3 for 2 months (Belyaeva, 1967) reproductive funtion was impaired with sterility and reduced numbers of viable offspring. These effects were reported to be the result of interference with ovigenesis. No abnormalities were reported in the offspring.

In a study in sheep (James et al, 1966) four ewes were given 2 mg/kg of antimony potassium tartrate orally in a gelatin capsule daily from the first day of gestation for 45 days (2 ewes) or 154-5 days (2 ewes). No adverse effects were noted in the ewes and all gave birth to normal full term lambs. No detectable amounts of Sb accumulated in the tissues of ewes on lambs.

h. Mutagenicity

No information available.

i. Carcinogenicity

In a lifetime study in mice (Kanisawa and Schroeder, 1969) white Swiss mice, Charles River strain (CD-1) were fed a diet low in trace metals and were given deionized drinking water to which the essential trace elements and 5 μg/ml antimony as the potassium tartrate had been added. No significant difference from the controls was noted in tumor incidence.

In a similar study in rats (Schroeder et al, 1970), random-bred Long Evans strain rats were given 5 ppm antimony as potassium antimony tartrate in drinking water. No increase in the incidence of tumors at any site was observed.

The Russian researchers, E.I. Erusalimskii and G.A. Suspa, publishing in Vop. Klin. Eksp. Onkol. 8: 239-42 (1972) on the "Deposition of 3,4-Benzopyrene in Rat Lungs Under the Influence of Metallic Antimony," reported that 5 mg of metallic antimony administered to rats along with 4 mg of 3,4-benzopyrene given intratracheally potentiated benzopyrene carcinogenicity in the rat lungs.

3. Effects on Other Vertebrates

Halstead (1972) discusses the effects of chemical intoxicants (including Sb) on the quality of marine products and possibly, as a consequence, on human health. He cites various examples of cancerous growths, leukemia, genetic changes and other effects of polluted waters on fish and shellfish, and mentions the current concern about human poisonings associated with eating contaminated seafoods. However, the likelihood of large concentrations of antimony in the marine environment is slight because the few antimony salts that are soluble tend to precipitate from solution as Sb_2O_3 or Sb_2O_5.

In discussing automated monitoring for marine pollution studies, Gafford (1972) suggests a classification scheme for minor elements with respect to marine pollution, placing antimony in the category of elements singled out for special interest because of their exceptional toxicity or that of some of their elements.

a. Bioaccumulation

Average concentrations of antimony in the oceans are estimated at 0.33 mg/l. According to Goldberg, E.D. (1957), "Biogeochemistry of Trace Metals," in Treatise on Marine Ecology and Paleacology, J.W. Hedgpeth, ed., Geological Society of America Memoir 67, vol.1, pp.345-57, and to Noddack, I. and Noddack, W. (1939), "Die Haufigkeit der Schwermetalle in Meerestieren," Arkiv. Zool. 32A (4): 1-35, antimony can be concentrated by certain marine forms (e.g., ascidian, sponge, acaleph, actinian, echinoid, asteroid) to over 300 times the amount present in sea water.

Leatherland and Burton (1974) reported that when antimony was detected in concentrations of 0.01 and 0.1 ppm in the bottom muds of waters near Southhampton, England, the values for fish muscle were at the lower end of the concentration range.

As mentioned on p. 121, when potassium antimony tartrate was added to the drinking water of rats at a concentration of 5 ppm for the lifetime of the animals (Schroeder *et al*, 1970), antimony accumulated in the tissues with age. In a similar study, increased levels of antimony were found in the tissues of mice (Schroeder *et al*, 1968) given 5 ppm of antimony potassium tartrate in their drinking water. Gross *et al* (1955b) reported an accumulation of antimony in the lung tissues of rats and rabbits exposed to air containing antimony trioxide (particle size about 0.6 μ) at concentrations of 100-125 ppm and 85 ppm, respectively for 10 hours/month for 14.5 months and 10 months, respectively.

b. Acute Toxicity

Antimony ions in soft water were toxic to fish (fathead minnows) at concentrations of 20 ppm. In hard water, the toxic concentration was somewhat lower. The ratio of concentration in hard:soft water was 0.6, essentially reducing the allowable concentration in hard water to 12 ppm. So reports Schroeder (1965), using data from McKee, J.E. and Wolf, H.E., editors, *Water Quality Criteria*, 2nd ed., State Water Quality Control Board, Sacramento, California, 1963.

Nine years later, apparently based on some of the same basic references used for the above, the National Academy of Sciences (NAS) reports on p. 243 of Water of Water Quality Criteria 1972 that although few antimonial salts have been tested on fish in bioassays, particularly in sea water, in static acute bioassays on fathead minnows (*Pimephales promelas*) antimony potassium tartrate gave a 96-hr LC_{50} as antimony of 20 mg/ℓ in soft water and 12 mg/ℓ in hard water; while antimony trichloride in similar tests gave a 96-hr LC_{50} as antimony of 9 mg/ℓ in soft water and 17 mg/ℓ in hard water.(1)

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(1) Tarzwell, C.M. and Henderson, C. (1960), "The Toxicity of Some of the Less Common Metals to Fishes," Transactions Seminar on Sanitary Engineering Aspects of the Atomic Energy Industry (Robert A. Taft Sanitary Engineering Center, TID-7517), and Tarzwell, C.M. and Henderson, C. (1960), "Toxicity of less Common Metals to Fishes," Indust. Wastes 5: 12.

Citing two other studies, the NAS also reports that at 5 mg/l neither $SbCl_3$ nor $SbCl_5$ had any effect on rainbow trout, bluegill sunfish, and sea lamprey in Lake Huron water at 13°C, saturated with dissolved oxygen, and pH 7.5 to 8.2,(1) but that projectile vomiting occurred in large mouth bass exposed to 1.0 mg/l of antimony as tartar emetic.(2)

c. Histochemistry

In a study to locate sites of ion transport, pieces of kidney from sea lamprey, Petromyzon marinus, were fixed in a solution of potassium pyroantimonate which has been used to detect cations in tissue. The cells of the proximal tubule apparently were the only epithelium within the kidneys of both larvae and adults which possessed electron-dense deposits of pyroantimonate within the cytoplasm of the cells and within the lateral intercellular spaces. All cells within the kidneys contained deposits in their nucleoplasm. The cells of the proximal tubule had large accumulations of deposit in membranous vesicles concentrated near the plasma membrane at all surfaces. With the exception of the Golgi apparatus little or no precipitate was observed within cell organelles and, therefore, there appeared to be little cell injury (Youson, 1973).

4. Effects on Invertebrates

Tissue Uptake - When host mice infected with an Egyptian strain of Schistosoma mansoni were given intraperitoneal infection of 5 mg/kg of potassium antimony tartrate, worms collected from the portal system showed uptake of antimony one hour after injection. The uptake of Sb was higher in females than in males but the range of concentrations was quite narrow. The highest concentrations were found 2 hours after injection. In males antimony concentrations were highest in worm segments containing testes suggesting accumulation in that organ. No similar apparent accumulation was found in the ovaries of females (Molokhia and Smith, 1968).

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(1) Applegate, V.C., J.H. Howell, A.E. Hall and M.A. Smith (1957), "Toxicity of 4,346 Chemicals to Larval Lampreys and Fishes," Fish and Wildlife Service, Special Science Report Fish 207: 157

(2) Jernejcic, F. (1969), "Use of Emetic to Collect Stomach Contents of Walleye and Large Mouth Bass," Transactions American Fisheries Society 98 (4): 698-702).

Leatherland and Burton (1974) reported that concentrations of 0.2 ppm antimony were found in two ascidians (sea squirts) in the bottom muds of waters near Southhampton, England. Ascidians are identified as a species having an outstanding capacity for concentrating such trace metals as vanadium.

Toxicity - On p. 243 of its Water Quality Criteria 1972, the NAS reports that (antimony) at 9 mg/l retarded the movement of *Daphnia*, as recorded by Bringmann, G. and R. Kuhn (1959), "The Toxic Effect of Waste water on Aquatic Bacteria, Algae, and Small Crustaceans," Gesundh. Ing. 80: 115-20.

In a study in mice infected with Schistosoma mansoni, administration of potassium antimony tartrate to the mice resulted in detectable damage to the reproductive organs of female worms within 5 minutes. This effect was reversed in the 5 months following treatment (Bourgeois, 1971).

Rogers and Howell (1971) have reported that antimony compounds administered to the fowl tick, Argas radiatus, by *in vivo* engorgement on poisoned chicks, *in vitro* engorgement on preserved chicken blood, lanolin inunctum or dipping of the ova in physiological saline containing antimony compounds, significantly reduced survival in larvae, neonymphs, deuternymphs and adults, fecundity in resulting females and fertility in deposited ova. Histologically, gametic destruction was observed in both sexes. Percent hatch was higher for ova dipped in saline containing potassium antimony tartrate than for those dipped in saline alone, but was lower than for untreated ova.

5. Effects on Plants

On p.243 of its Water Quality Criteria 1972, the NAS again cites Bringmann and Kuhn (ibid) as reporting that (antimony) at 3.5 mg/l hindered the cellular division of green algae.

6. Effects on Microorganisms

a. Toxicity and Pharmacology

In a study using five strains of Staphylococcus aureus (phage types 7 US, 54, 71, 80 and 81) all of which were of human origin, hemolytic, coagulase positive, and penicillin resistant neither antimony sodium thioglycollate or antimony thioglycollamide at concentrations up to 0.125 ug/ml had any affect on the growth of the bacteria during an 18 hour incubation period (Richtarik et al, 1965).

With Eschericia coli, B bacteriostatic concentrations of piperazine di-antimony tartrate (bilharzid) and potassium antimony tartrate (tartar emetic) were 4.244 x 10^{-6} M/ml and 19.510 x 10^{-6} M/ml, respectively. On removal of the antimonial drug from the medium, cell division was re-initiated and proceeded similarly to controls. With each drug at the bacteriostatic concentration there was almost complete inhibition of the synthesis of DNA and RNA. Protein synthesis was completely inhibited for the first two hours followed by a low rate production for the next two hours. Removal of the inhibitor resulted in resumption of the biosynthetic processes (Khafagy and El-Hawary, 1974).

Formation of trimethylarsine from sodium arsenate by Candida humicola was not affected by low concentrations (0.025% and 0.050%) of sodium antimonate ($NaSbO_3$), but was reduced by a concentration of 0.10% $NaSbO_3$ (Cox and Alexander, 1973).

Mutagenesis - The differential growth sensitivities to metal compounds in wild and recombination deficient strains of Bacillus subtilis were tested using the strains H17 (Rec^+, arg^- and trp^-) and M45 (Rec^-, arg^- and trp^-). The difference between the inhibition zones produced by the compounds for Rec^+ and Rec^- is called the "rec-effect". It is suggested that rec-assay positive compounds are mutagens. Antimony chloride did not give positive effects in the assay (Nishioka, 1975).

7. In Vitro and Biochemical Studies

a. Effects on Isolated Organs

Mouse liver slices incubated with ^{124}Sb-labelled antimony potassium tartrate accumulated antimony establishing a concentration gradient. With a concentration of 10^{-5}M tissue:medium ratios rose to a mean of 38.3:1 indicating very powerful concentrating ability. After 2 hours no further uptake occurred. With prolonged incubation the liver slices deteriorated with dead-looking tissue sloughing off. This tissue, which microscopically appeared as undifferentiated necrotic tissue, contained 3 to 18 times as much antimony as healthy tissue (Smith, 1969).

Subcellular fractionation of the liver slices following incubation with ^{124}Sb-antimony potassium tartrate resulted in about 70% of the radioactivity being recovered in the particulate fractions. The microsomal fraction contained the highest concentrations in terms of both protein and tissue weight (Smith, 1969).

Antimony uptake by liver slices incubated 1-8 minutes was unaffected by absence of oxygen, presence of KCl, oubain, dinitrophenol or Na arsenate, or by preincubation with unlabelled antimony potassium tartrate for up to 60 minutes. Uptake was inhibited by dimercaprol, dithiothreitol and Ellman's reagent. Uptake of antimony was associated with progressive reductions in oxygen consumption (Smith, 1969).

Slices of other tissue were able to take up antimony but to a lesser extent than liver. Accumulating ability decreased in order liver > kidney > spleen > intestine > brain (Smith, 1969).

In a study of cardiac effects potassium antimony tartrate injected into the coronary circulation of isolated spontaneously beating dog hearts produced a progressive fall in myocardial contractile force which was not reversible. Some bradycardia was observed but the degree was neither consistent or remarkable. Post-stimulation potentiation of contractile force which is usually seen was absent in a number of instances. However, the magnitude of the percentage response to test doses of adrenalin was essentially unchanged by antimony (Bromberger-Barnea and Stephens, 1965).

In isolated spontaneously beating guinea pig atria increasing concentrations of potassium antimony tartrate (tartar emetic) and sodium antimony dimercaptosuccinate (TWSb) had only a slight effect upon rate until a concentration of 10^{-2}M was reached. At this concentration both antimonials caused substantial and significant reduction in contractile force. Similar responses were observed in guinea pig ventricle strip preparations (Cotton and Logan, 1966).

b. Effects on Cell Cultures

Incubation of horse blood with trivalent antimony as antimony potassium tartrate (tartar emetic) or sodium antimony dimercaptosuccinate (Astiban) resulted in increasing uptake of Sb by erythrocytes with time up to 8 hours. With pentavalent antimony as sodium stibogluconate (Pentostam) very little uptake occurred regardless of incubation time. At all times plasma concentrations were much higher than erythrocyte concentrations (Molokhia and Smith, 1969b).

Erythrocytes labelled with trivalent antimony and incubated with normal plasma for 1 hour lost antimony to the plasma. This was slightly more pronounced with tartar emetic than with Astiban. More antimony was lost from cells which had been incubated shorter periods during labelling (Molokhia and Smith, 1969b).

In cultured human leukocytes, sodium antimony tartrate at a concentration of 1.0×10^{-8}M added to the culture during the last 48 hours of a 72 hour culture period was toxic to the cells, causing a marked reduction in mitotic index. For chromosome studies a concentration of 2.3×10^{-9}M was added to the leukocyte culture medium. Of a total of 100 metaphases examined, 12% of the cells had chromatid breaks which was a statistically significant (5% level) increase over the controls (Paton and Allison, 1972).

In human blood cells selective binding of pyroantimonate was observed in the plasmalemma of developing and mature neutrophilic leukocytes, but not in other forms of leukocytes. In the earliest stages of development the plasmalemma is unreactive. Binding was first evident in young forms of the midpromyelocyte and reached a peak during the latter half of the promyelocytic stage. In the segmented neutrophil there were only a few deposits of reactive product associated with the plasmalemma. Late developmental forms of nucleated erythrocytic cells and mature erythrocytes also exhibited plasmalemmal pyroantimonate binding. The degree of binding was related to the degree of maturation. These results indicate that chemical distinctions between the plasmalemma of different types of cells exist (Ackerman and Clark, 1972).

c. Effects on Isolated Organelles and Cell Homogenates

Rat liver mitochondria pre-incubated for 2 minutes with increasing amounts of sodium antimony gluconate (Triostib, 30% trivalent antimony) showed progressive inhibition of oxygen when α-ketoglutarate was the substrate indicating that oxidative phosphorylation was increasingly inhibited. The same result was observed when the substrate was glutamate, but not when it was succinate. Similar results were obtained in classical Warburg experiments. Since it was the oxidation of NAD^+ linked substrates that was inhibited the investigators assumed that Triostib probably acted on the NADH-oxidase segment of the chain. The inhibition of NADH oxidation was reversed by the addition of methylene blue (Campello *et al* 1970).

d. Effects on Enzyme Systems

In extracts from homogenized adult *Schistosoma mansoni* worms, lactic acid production from glucose was markedly inhibited when stibophen or antimony potassium tartrate was added. Both purified and crude glucokinase preparations were relatively insensitive to high concentrations of antimonials indicating that phosphorylation of glucose to glucose-6-phosphate (G-6-P) by ATP was not affected by Sb. Fructokinase and mannokinase were also not affected. Neither antimony compound affected conversion of G-6-P to fructose-6-phosphate (F-6-P) or lactic production from fructose-1, 6-diphosphate (HDP). However, lactic acid production from F-6-P was inhibited indicating that the antimonials block the formation of HDP from F-6-P by the enzyme phosphofructokinase. (Mansour and Bueding, 1954). In rat brain preparations, phosphofructokinase activity was much less sensitive to the trivalent antimony compounds. Pentavalent antimony as sodium stibogluconate had no effect on the activity of the worm enzyme.

e. Effects on Nucleic Acids and Proteins

There was no information in the literature.

IV. REGULATIONS AND STANDARDS

A. Current Regulations

1. Food and Drug Authorities

The proportion of antimony in color additives is limited, according to Part 8 of the CFR Title 21, Chapter 1 - Food and Drug Administration (FDA). In Subpart D, the 8.300 series concerns diluents in color additive mixtures for food use exempt from certification, under which specifications for antimony are listed in 8.316. This section states that the synthetically prepared color additive titanium dioxide (TiO_2) shall conform to the following:

Lead (as Pb), not more than 10 parts per million.
Arsenic (as As), not more than 1 part per million.
Antimony (as Sb), not more than 2 parts per million.
Mercury (as Hg), not more than 1 part per million.
Loss on ignition at 800°C. (after drying for 3 hours at 105°C), not more than 0.5 percent.
Water soluble substances, not more than 0.3 percent.
Acid soluble substances, not more than 0.5 percent.
TiO_2 not less than 99.0 percent after drying for 3 hours at 105°C.

2. Air and Water Limitations

The release into the environment of antimony and of such compounds of antimony as antimony trioxide, antimony pentasulfide, antimony sulfate, antimony trisulfide, antimony trifluoride, antimony pentafluoride and potassium antimony tartrate is provisionally limited to contamination levels of 0.005 ppm (mg/l) as Sb in air and 0.05 ppm (mg/l) as Sb in water (TRW, 1973). These levels based on threshold limit values, were calculated by TRW in a study for EPA on recommended methods for disposal of hazardous waste. They are offered as provisional limiting concentrations pending the development of ambient air quality standards that can be based on extensive evaluation of acute and chronic dose response criteria for exposure of all living and nonliving things.

The U.S. Environmental Protection agency in October 1975 issued its "Development Document for Interim Final and Proposed Effluent Limitations Guidelines and New Source Performance Standards for the Ore Mining and Dressing Industry" (EPA 440/1-75/061, Vols. I and II). On p. 753 of Vol. II, the effluent limitation for Sb (recommended for best practicable control technology currently available (BPCTCA) -- antimony mines) is 0.5 mg/ℓ Sb for a 30-day average and 1.0 mg/ℓ for a 24-hour maximum.

The EPA's National Interim Primary Drinking Water Regulations, published in the Federal Register of 24 December 1975, are to be effective 24 June 1977. These regulations do not include antimony among the inorganic chemicals for which maximum contaminant levels are given.

3. Wastes

Antimony pentafluoride and antimony trifluoride are listed as nonradioactive High hazard compounds by the EPA Office of Solid Waste Management Programs in its 1974 Report to the Congress on the Disposal of Hazardous Wastes, identified as EPA Publication SW-115. The Office of Solid Waste Management Programs considers these two compounds of antimony unacceptable for normal disposal methods.

4. Occupational Safety and Health Administration (OSHA)

Under the Occupational Safety and Health Standards, announced in 1974 by OSHA, antimony and compounds (as Sb) are listed together as one entry in a table of air contaminants for which employee exposure limits are given as 8-hour time weighted averages. According to the OSHA standards, an employee's exposure to antimony and compounds (as Sb) shall not exceed the 8-hour time weighted average of 0.5 mg/m^3, for any 8-hour work shift of a 40-hour week. [Fed. Reg. 39 (125), p.23540-1 (27 June 1974)]

5. Transport Regulations

In the CFR 46 for Shipping, Chapter I - Coast Guard, Department of Transportation, only antimony pentachloride and antimony pentafluoride are included on the list of explosives and other dangerous articles and combustible liquids. Antimony pentachloride may be shipped dry or in solution but by cargo vessel only. It should be stored on deck, protected from the weather or under cover and carry a white label for corrosive liquid. Transport limits are given as follows:

- for metal barrels or drums - 55 or 110 gallon capacity
- for wooden barrels - 20016 net weight
- for wooden boxes - 20016 gross weight
- for portable tanks - 8,00016 gross weight
- for tank cars - must comply with DOT regulations (trailerships only)
- for motor vehicle tank trucks - must comply with DOT regulations (trailerships and trainships only)

The regulations for antimony pentachloride solution are the same as for $SbCl_5$ in dry form except for very detailed specifications for containers.

Antimony pentafluoride is permitted to be transported by cargo vessel only in steel cylinders containing 1, 5, 25 and 200 lb SbF_5 and stored on deck protected from the weather or under cover. White labels are required signifying corrosive liquid. Electric wet storage batteries containing SbF_5 are listed by DOT.

6. Other Federal

The U.S. Bureau of Mines has approved various types of respirators for personal protection against antimony contamination, depending on the form of contaminant.

7. States

a. New Jersey

Chapter VII of the Code of New Jersey Air Pollution Control Commission, New Jersey State Department of Health covers air pollution from solid particles. Under Section 2.7 of this code antimony is listed with an "effect factor" of 0.9 for fine solid particles. An effect factor in this context is defined as a value assigned to an air contaminant in the form of a numerical modifier which when multiplied by the basic emission yields the allowable emission.

b. Pennsylvania

The Hygienic Information Guide No. 11 of Pennsylvania Department of Health, Division of Occupational Health includes antimony. A Threshold Limit-Value (acceptable atmospheric concentration for 8-hour work period) of 0.1 ppm of air is given for stibine and 0.5 mg/m^3 of air for antimony and its compounds (as Sb). This Guide states that inhalation of gases, fumes and dusts of antimony and its compounds is the main source of antimony poisoning and exposure to stibine concentrations of 100 ppm for several hours may cause death.

Recommended exposure control includes maintaining atmospheric contamination levels at less than Threshold Limit Value by enclosure and local exhaust ventilation. Personal respiratory protection, approved by the U.S. Bureau of Mines for protection against the form of antimony contamination generated, is suggested. For prevention of skin contact suitable gloves, aprons and fact shields should be used.

8. Foreign

The Inter-Governmental Maritime Consultative Organization (IMCO) lists antimony lactate, antimony potassium tartrate, and antimony (inorganic, not otherwise specified) in its International Maritime Dangerous Goods Code. However, the provisions of this Code should not apply to antimony sulfides and oxides free of arsenic.

Antimony compounds, including antimony lactate and antimony potassium tartrate, are classified as poisonous (toxic) substances which are liable to cause death, or serious injury to human health if swallowed, inhaled, or by skin contact. The container and package regulations are essentially the same for the antimony compounds designated and a poison label must be displayed. Size limits are stipulated for liquid and solid form and vary depending upon whether the packing mode is glass bottles or cans, or (in the case of solids only) paper or plastic bags in a wooden or fibreboard box; a metal drum; or (in the case of solids only) a wooden barrel, fibre or plywood drum. Storage on deck or under deck is required with fibreboard boxes, in particular, stowed under deck and unexposed to sea water. All storage is limited to cargo ships or passenger ships, which are carrying not more than 25 passengers or 1 per 10 feet of length, and separated from all foodstuffs in order to avoid contamination.

One study (Arzamastsev, 1964), performed at a Soviet institute for community health, resulted in recommendations that the maximum permissible concentration of trivalent and pentavalent antimony in water be set at 0.05 mg/l. This investigation showed 0.0025 mg/kg had no effect on warm-blooded animals and 0.6 mg/l as the threshold values affecting organoleptic properties of water for both trivalent and pentavalent antimony. The LD_{50} values for albino rats amounted to 675 mg/kg for $SbCl_3$ and 1115 mg/kg for $SbCl_5$.

B. Consensus and Similar Standards

The American Conference of Governmental Industrial Hygienists (ACGIH) has adopted 0.5 mg/m^3 as the Threshold Limit Value (TLV) for airborne concentrations of antimony and compounds (as Sb). This TLV represents conditions under which it is believed that nearly all workers may be repeatedly exposed day after day without adverse effect.

V. EVALUATION AND COMMENTS

Antimony has been manufactured in the U.S. for about 80 years. Although there have been reported incidences of pneumoconiosis among workers, and antimony compounds can be irritating to the skin and cause other disorders, no epidemiological studies have appeared. There is no evidence that antimony is a carcinogen, teratogen, or a mutagen.

A limited number of manufacturers produce antimony and/or its compounds and production is comparatively small scale, being only about 41,000 tons/year.

The most serious potential problems associated with antimony wastes seem to be related with their potential impact on the ecosystem, resulting from disposal in fresh or sea waters. To date, however, no evidence exists to indicate that antimony or its compounds in the amounts detected in water have had any harmful effects on marine life.

While current knowledge does not permit ranking antimony and its compounds as major environmental contaminants, antimony has innate toxicity and inhalation of concentrations above its Threshold Limit Value could be damaging. Careful control of antimony air emissions, careful monitoring of antimony-containing effluents, and proper disposal of toxic antimony compounds or those antimony compounds that degrade to form toxic substances should be encouraged.

REFERENCES

Abdallah, Ahmed and Ahmed Badran (1961), EFFECT ON THE ELECTROCARDIOGRAM OF POTASSIUM ANTIMONY TARTRATE (TARTAR EMETIC) IN PATIENTS TREATED FOR SCHISTOSOMIASIS. J. Egypt. Med. Assoc. 44: 767-71

Abdallah, A. and M. Saif (1962), TRACER STUDIES WITH ANTIMONY-124 IN MAN. in: Bilharziasis, G.E.W. Wolstenholme and M. O'Connor, Eds., Little, Brown & Co., Boston, pp 287-309

Abdallah, A. and A. Badran (1963), EFFECT OF ANTIMONY DIMERCAPTOSUCCINATE ON THE ELECTROCARDIOGRAM IN PATIENTS TREATED FOR SCHISTOSOMIASIS. Am. J. Trop. Med. Hyg., 12: 188-92

Ackerman, G.A. and M.A. Clark (1972), A CYTOCHEMICAL EVALUATION OF PYROANTIMONATE BINDING TO THE PLASMALEMMA OF BLOOD AND BONE MARROW CELLS AND ITS RELATIONSHIP TO CELLULAR MATURATION. J. Histochem. Cytochem. 20 (11): 880-95

Arzamastsev, E.V. (1964), MAXIMUM PERMISSIBLE CONCENTRATION OF TRI- AND PENTAVELENT ANTIMONY IN SUPPLIES. Gigiena i Sanit. 29 (12): 16-20 (in Russian)

Asarco (1975), NEWS RELEASE (31 October)

Baetjer, A.M. (1969), EFFECTS OF DEHYDRATION AND ENVIRONMENTAL TEMPERATURE ON ANTIMONY TOXICITY. Arch. Environ. Health 19 (6): 784-92

Baetjer, A.M. (1973), DEHYDRATION AND SUSCEPTIBILITY TO TOXIC CHEMICALS. Arch. Environ. Health 26: 61-3

Bahner, C.T. (1954), LOCALIZATION OF ANTIMONY IN BLOOD. Proc. Soc. Exp. Biol. Med. 86: 371-73

Barr, L. (1975), Private Communication With V. Valeri, ADL, (13 November)

Bartlet, J.C. and J.L. Monkman (1964), SUMMARY OF RESULTS OF THE COLLABORATIVE TESTS ON THE RHODAMINE B COLORIMETRIC METHOD FOR ANTIMONY. J. of the A.O.A.C. 47 (4): 630-2 (August)

Belcher, R., S.L. Bogdanski, S.A. Ghonaim and A. Townshend (1974), MOLECULAR EMISSION CAVITY ANALYSIS(MECA)--A NEW FLAME ANALYTICAL TECHNIQUE. PART IV. THE DETERMINATION OF ARSENIC AND ANTIMONY. Analytica Chimica Acta 72 (1): 183-7

Belyaeva, A.P. (1967), THE EFFECT PRODUCED BY ANTIMONY ON THE GENERATIVE FUNCTION. Gigiena Truda i Professional-nye Zabolevaniia 11 (1): 32-7

Bloch, P. (1970), GAMMA RAY SPECTROMETRIC METHOD USING A LITHIUM DRIFTED SILICON DETECTOR TO DETERMINE THE ANTIMONY DEPOSITS IN THE LUNG IN VIVO. Radiology 96 (3): 657-8

Booz-Allen Applied Research Inc. (1972), ANTIMONY. in: A Study of hazardous of Waste Materials; Hazardous Effects and Disposal Methods, Vol. 2., Appendix A-1, pp 98-100 (30 June)

Bou Casals, J. (1972), PHARMACOKINETIC AND TOXICOLOGICAL STUDIES OF ANTIMONY DEXTRAN GLYCOSIDE (RL-712). Br. J. Pharmac. 46 (2): 281-8

Bourgeois, John G. (1971), FURTHER EVALUATION OF AN INTRA VITAM STAINING METHOD FOR THE DETECTION OF EARLY IN VIVO ANTISCHISTOSOMAL EFFECTS. J. Exp. Pharmacol. Exp. Ther. 176 (2): 455-63

Brieger, H., C.W. Semisch, J. Stasney and D.A. Piatnek (1954), INDUSTRIAL ANTIMONY POISONING. Ind. Med. Surg. 23: 521-3

Bromberger-Barnea, B. and N.L. Stephens (1965), EFFECTS OF ANTIMONY ON MYOCARDIAL PERFORMANCE IN ISOLATED AND INTACT CANINE HEARTS. Am. Ind. Hyg. Assoc. J. 26 (4): 404-8

Brune, D., K. Samsahl and P.O. Wester (1963), DETERMINATION OF ELEMENTS IN MILLI-, MICRO- AND SUBMICROGRAM QUANTITIES IN HUMAN WHOLE BLOOD BY NEUTRON ACTIVATION ANALYSIS. Atompraxis 9: 368-73

Bulmer, F.M.R. and J.H. Johnston (1948), ANTIMONY TRISULFIDE. J. Ind. Hyg. and Toxicol. 30 (1): 26-8

Burgess, A.E. and J.M. Ottaway (1972), A KINETIC METHOD FOR THE DETERMINATION OF ARSENIC (III), ANTIMONY (III) AND ASCORBIC ACID. Analyst 97 (1154): 357-62 (May)

Campello, A.P., D. Brandao, M. Baranski and D.O. Voss (1970), STUDIES OF SCHISTOSOMICIDES ANTIMONIALS ON ISOLATED MITOCHONDRIA --I. SODIUM ANTIMONY GLUCONATE (TRIOSTIB). Biochem. Pharmac. 19 (5): 1615-19

Casey, James P. (1966), in: Pulp and Paper Chemistry and Chemical Technology, 2nd Ed., Volume III: Paper Testing and Converting, Interscience Publishers, Div. of John Wiley and Sons, p. 1587

Chekunova, M.P. and N.A. Minkina (1970), AN INVESTIGATION OF THE TOXIC EFFECT OF ANTIMONY PENTAFLUORIDE. Hygiene & Sanitation 35 (7): 30-4

Chem. Eng. News (1971), TRACE METALS: UNKNOWN, UNSEEN POLLUTION THREAT. 49 (29): 29-30, 33 (19 July)

Chem. Eng. News (1974), Du PONT DRAWS FIRE ON GULF DUMPING. 52 (30): 5 (29 July)

Chem. Mkt. Rpt. (1975), ANTIMONY OXIDE DEMAND STIRS? PRICES ARE CUT FOR SECOND TIME. 207 (15): 31 (14 April)

Chem. Wk. (1973), ANTIMONY OXIDE MARKETS APPEAR HEADED FOR SUBSTANTIAL GROWTH. p 19 (31 January)

Chusid, J.G. and L.M. Kopeloff (1962), EPILEPTOGENIC EFFECTS OF PURE METALS IMPLANTED IN MOTOR CORTEX OF MONKEYS. J. Appl. Physiol. 17 (4): 697-700

Clarke, E.G.C. (1963), A MODIFIED APPARATUS FOR REINSCH'S TEST. J. Pharm. Pharmacol. 15 (1): 73-5

Cooper, D.A., E.P. Pendergrass, A.J. Vorwald, R.L. Maylock and H. Brieger (1968), PNEUMOCONIOSIS AMONG WORKERS IN AN ANTIMONY INDUSTRY. Am. J. Roentgenol. Radium Ther. Nucl. Med. 103 (3): 495-508

Cordasco, E.M. and F.D. Stone (1973), PULMONARY EDEMA OF ENVIRONMENTAL ORIGIN. Chest 64 (2): 182-85

Cotten, M. DeV. and M.E. Logan (1966), EFFECTS OF ANTIMONY ON THE CARDIOVASCULAR SYSTEM AND INTESTINAL SMOOTH MUSCLE. J. Pharmacol. Exp. Therap. 151 (1): 7-22

Cox, D.P. and M. Alexander (1973), EFFECT OF PHOSPHATE AND OTHER ANIONS ON TRIMETHYLARSINE FORMATION BY CANDIDA HUMICOLA. Appl. Microb. 25 (3): 408-13

Crecelius, E.A., C.J. Johnson and G.C. Hofer (1974), CONTAMINATION OF SOILS NEAR A COPPER SMELTER BY ARSENIC, ANTIMONY AND LEAD. Water, Air and Soil Pollution 3 (3): 337-42

Crecelius, Eric A., Michael H. Bothner and Roy Carpenter (1975), GEOCHEMISTRIES OF ARSENIC, ANTIMONY, MERCURY, AND RELATED ELEMENTS IN SEDIMENTS OF PUGET SOUND. Environ. Sci. & Tech. 9 (4): 325-33

Davis, A. (1961), THE EFFECT OF ANTIMONY DIMERCAPTO-SUCCINATE ON THE ELECTROCARDIOGRAM. Br. Heart J. 23: 291-96

Davison, Richard L., David F.S. Natusch, John R. Wallace, Charles A. Evans, Jr. (1974), TRACE ELEMENTS IN FLY ASH -- DEPENDENCE OF CONCENTRATION ON PARTICLE SIZE. Environ. Sci. & Tech. 8 (13): 1107-13 (December)

Dubois, L. and J.L. Monkman (1964), POLAROGRAPHIC DETERMINATION OF HEAVY METALS IN AIR SAMPLES. Am. Ind. Hyg. Assoc. J. 25 (5): 485-91 (Sept/Oct)

El-Hawey, A.M., M.S. Ibrahim, A.H. Mousa, and H.A. Ghaleb (1971), FURTHER EXPERIMENTAL STUDIES ON THE ACTION OF ANTIMONIALS AND SCHISTOSOMA MANSONI WORM ANTIGEN ON THE TRACHEOBRONCHIAL TREE. J. Egypt. Med. Assoc. 54: 502-7

EPA (1974), WATERBORNE WASTES OF THE PAINT AND INORGANIC PIGMENTS INDUSTRIES. p 40 (March) PB-232 019

Ercoli, N. (1968), CHEMOTHERAPEUTIC AND TOXICOLOGICAL PROPERTIES OF ANTIMONYL TARTRATE-DIMETHYLCYSTEINE CHELATES (33304). Proc. Soc. Exper. Biol. Med. 129 (1): 284-90

Ercoli, N. (1971), SIGNIFICANCE OF THE CHEMOTHERAPEUTIC INDEX IN THE TREATMENT OF SCHISTOSOMIASIS WITH ANTIMONY COMPOUNDS. Bull. WHO 45 (3): 371-5

Feigl, F. and F.L. Chan (1967a), SPOTS TESTS FOR THE DETECTION OF ELEMENTAL ARSENIC, ANTIMONY, AND TIN. Chemist Analyst 56 (1-2): 13-14 (January)

Feigl, F. and F.L. Chan (1967b), SPOT TESTS FOR THE DETECTION OF BOUND ARSENIC AND ANTIMONY. Chemist-Analyst 56 (1-2): 14 (January)

Feigl, F. and V. Anger (1972), ANTIMONY. in: Spot Tests in Inorganic Analysis, Elsevier Publishing Co., N.Y., pp 104-11

Felicetti, S. W., R.G. Thomas and R.O. McClellan (1974a), METABOLISM OF TWO VALENCE STATES OF INHALED ANTIMONY IN HAMSTERS. Amer. Ind. Hyg. Assoc. J. 35 (5): 292-300

Felicetti, S.W., R.G. Thomas and R.O. McClellan (1974b), RETENTION OF INHALED ANTIMONY-124 IN THE BEAGLE DOG AS A FUNCTION OF TEMPERATURE OF AEROSOL FORMATION. Health Phys. 26: 525-31

Filer, T.D. (1971), FLUOROMETRIC DETERMINATION OF SUBMICROGRAM QUANTITIES OF ANTIMONY. Analytical Chemistry 43 (6): 725-9 (May)

Finney, Joseph W. (1975), ANTIMONY -- DEFLATED MARKETS LOOK TO OXIDE FOR GROWTH. Engineering and Mining Journal 176 (3): 198-9 (March)

Gafford, R.D. (1972), AUTOMATION OF MONITORING EQUIPMENT FOR MARINE POLLUTION STUDIES. in: Marine Pollution and Sea Life, Mario Ruivo, Ed., Fishing News (Books) Ltd., Surrey & London, England, pp 491-500

Gilbert, Thomas R. and David N. Hume (1973), DIRECT DETERMINATION OF BISMUTH AND ANTIMONY IN SEA WATER BY ANODIC STRIPPING VOLTAMMETRY. Analytica Chimica Acta 65 (2): 451-9

Girgis, G.R., P. Scott, A.R. Schulert and H.G. Browne (1965), ACUTE TOLERANCE OF MICE TO TARTAR EMETIC. Toxicol. Appl. Pharmacol. 7: 727-31

Goulden, P.D. and Peter Brooksbank (1974), AUTOMATED ATOMIC ABSORPTION DETERMINATION OF ARSENIC, ANTIMONY, AND SELENIUM IN NATURAL WATERS. Anal. Chem. 46 (11): 1431-6

Gross, P., J.H.U. Brown, M.L. Westrick, R.P. Srsic, N.L. Butler and T.F. Hatch (1955a) TOXICOLOGIC STUDY OF CALCIUM HALOPHOSPHATE PHOSPHORS AND ANTIMONY TRIOXIDE. I. ACUTE AND CHRONIC TOXICITY AND SOME PHARMACOLOGIC ASPECTS. Arch. Indus. Health 11: 473-8

Gross, P., M.L. Westrick, J.H.U. Brown, R.P. Srsic, H.H. Schrenk and T.F. Hatch (1955b), TOXICOLOGIC STUDY OF CALCIUM HALOPHOSPHATE PHOSPHORS AND ANTIMONY TRIOXIDE. II. PULMONARY STUDIES. Arch. Indus. Health 11: 479-86

Gross, P., R.T.P. deTreville, E.B. Tolker, M. Kaschak and M.A. Babyak (1969), THE PULMONARY MACROPHAGE. Arch. Environ. Health 18: 174-85

Halstead, B.W. (1972), TOXICITY OF MARINE ORGANISMS CAUSED BY POLLUTANTS. in: Marine Pollution and Sea Life, Mario Ruivo, Ed., Fishing News (Books) Ltd., Surrey & London, England, pp 584-94

Harvey, S.C. (1960), ANTIMONY. in: The Pharmacological Basis of Thera-Peutics, 2nd Ed., L.S. Goodman and A. Gilman, Eds., The Macmillan Co., N.Y., pp 973-8

Hock, A., U. Demmel, H. Schicha, K. Kasperek and L.E. Feinendegen (1975), TRACE ELEMENT CONCENTRATION IN HUMAN BRAIN. Brain 98 (Part I): 49-64 (March)

Hoffman, I. (1969), REPORT ON METALS AND OTHER ELEMENTS. J. Assoc. Offic. Anal. Chem. 52 (2): 274-5 (March)

Hopps, Howard C. (1974), THE BIOLOGIC BASES FOR USING HAIR AND NAIL FOR ANALYSES OF TRACE ELEMENTS. in: Trace Substances in Environmental Health. VIII. Proc. of Univ. of Missouri's 8th Ann. Conf. on Trace Substances in Environ. Health, Columbia, Mo., pp 59-73 (11, 12, 13, June)

Hornedo, C.N. (1975), Private Communication with V. Valeri, ADL (13 November)

Hornedo, C.N. (1976), Private Communication with V. Valeri, ADL (8 January)

Howie, R.A., M.M. Molokhia, and Hamilton Smith (1965), ESTIMATION OF ANTIMONY IN BIOLOGICAL MATERIALS BY NEUTRON ACTIVATION ANALYSIS. 37 (8): 1059-61 (July)

James, L.F., V.A. Lazar and W. Binns (1966), EFFECTS OF SUBLETHAL DOSES OF CERTAIN MINERALS ON PREGNANT EWES AND FETAL DEVELOPMENT. Am. J. Vet. Res. 27 (1): 132-5

Jungers, Robert H., Robert E. Lee, Jr. and Darryl J. von Lehmden (1975), THE EPA NATIONAL FUELS SURVEILLANCE NETWORK. I. TRACE CONSTITUENTS IN GASOLINE AND COMMERCIAL GASOLINE FUEL ADDITIVES. Environ. Health Perspectives 10: 143-50 (April)

Kanisawa, M. and H.A. Schroeder (1969), LIFE-TERM STUDIES ON THE EFFECT OF TRACE ELEMENTS ON SPONTANEOUS TUMORS IN MICE AND RATS. Cancer Res. 29: 892-5

Karajovic, D. (1958), PNEUMOCONIOSES AMONG WORKERS IN AN ANTIMONY SMELTING PLANT. in: Proc. of XII International Congress on Occupational Health, Helsinki

Kennedy, J.H. (1966), ANALYSIS OF DISEASED AND NORMAL LUNG TISSUE FOR TRACE ANTIMONY CONTENT BY NEUTRON ACTIVATION ANALYSIS. Am. J. Med. Sci., pp 75-7

Khafagy, E.Z. and M.F. El-Hawary (1974), EFFECT OF BILHARCID AND TARTAR EMETIC ON DNA, RNA AND PROTEIN SYNTHESIS IN ESCHERICHIA COLI B. Biochem. Pharmacol. 23 (1): 1451-55

Kinser, Richard E., Robert G. Keenan, and Richard E. Kupel (1965), SPECTROCHEMICAL DETERMINATION IF INDIUM AND ANTIMONY IN BIOLOGICAL MATERIALS. Am. Ind. Hygiene Assoc. J. 26 (3): 249-54 (May-June)

Kirk-Othmer Encyclopedia of Chemical Technology (1963), ANTIMONY AND ANTIMONY ALLOYS. Volume 2, John Wiley & Sons, Inc., N.Y., pp. 562-88

Leatherland, T.M. and J.D. Burton (1974), THE OCCURRENCE OF SOME TRACE METALS IN COASTAL ORGANISMS WITH PARTICULAR REFERENCE TO THE SOLENT REGION. J. Mar. Biol. Assoc. U.K. 54 (2): 457-368

LeGall, (1969), PNEUMOCONIOSES ET ANTIMOINE. Arch. des Malades Profesionalles 30 (6): 361-62

Loh, C.L. and Y.R. Cie (1964), MICRODETERMINATION OF ANTIMONY IN BIOLOGICAL MATERIALS WITH BRILLIANT GREEN. Acta Pharm. Sin. 11 (7): 491-6 (in Chinese)

Mahmoud, A.A.F. and K.S. Warren (1974), ANTI-INFLAMMATORY EFFECTS OF TARTAR EMETIC AND NIRIDAZOLE: SUPPRESSION OF SCHISTOSOME EGG GRANULOMA. J. Immunology 112 (1): 222-228

Mansour, M.M., A.A.A. Rassoul, and A.R. Schulert (1967), ANTI-BILHARZIAL ANTIMONY DRUGS. Nature 214 (5090): 819-20 (20 May)

Mansour, T.E and E. Bueding (1954), THE ACTIONS OF ANTIMONIALS ON GLYCOLYTIC ENZYMES OF SCHISTOSOMA MANSONI. Br. J. Pharmacol. 9: 459-62

Markham, M. Clare, M. Consilia Hannan, Lily Lin, Claire Coffey, and Barbara Jones (1958), PHOTOCHEMICAL PROPERTIES OF ANTIMONY TRIOXIDE. J. Phys. Chem. 62 (8): 989-92 (3 September)

Mathews, C. M. E. and G. Molinaro.(1963) A STUDY OF THE RELATIVE VALUE OF RADIOACTIVE SUBSTANCES USED FOR BRAIN TUMOUR LOCALISATION AND OF THE MECHANISM OF TUMOUR: BRAIN CONCENTRATION, UPTAKE IN TRANSPLANTABLE FIBROSARCOMA, BRAIN AND OTHER ORGANS IN THE RAT. Br. J. Exp. Pathol. 44 (3): 260-277

Maxfield, D.J. M. Rodriguez, M. Buettner, J. Davis, L. Forbes, R. Kovacs, W. Russel, L. Schultz, R. Smith, J. Stanton and C.M. Wai (1974), HEAVY METAL POLLUTION IN THE SEDIMENTS OF THE COEUR D'ALENE RIVER DELTA. Environ. Pollut. 7 (1): 1-6 (July)

McCallum, R.I. (1963), THE WORK OF AN OCCUPATIONAL HYGIENE SERVICE IN ENVIRONMENTAL CONTROL. Ann. Occup. Hyg. 6 (2): 55-64

McCallum, R.I. (1967), DETECTION OF ANTIMONY IN PROCESS WORKERS' LUNG BY X-RADIATION. Trans. Soc. Occup. Med. 17 (4): 134-38

McCallum, R.I., M.J. Day, J. Underhill, and E.G.A. Aird (1970), MEASUREMENT OF ANTIMONY OXIDE DUST IN HUMAN LUNG *IN VIVO* BY X-RAY SPECTROPHOTOMETRY. British Occupational Hygiene Society, London, Inhaled particles III, Proc. Int. Symp., London, England (1970)

Miller, M.H. (1973), ANTIMONY. in: United States Mineral Resources, Brobst, D.A. and W.P. Pratt, Editors, U.S. GPO, Washington, D.C., pp 55-9

Mod. Plastics (1975a), THERE ARE NEW WAYS TO TAME THE FLAME 52 (5): 42-5 (May)

Mod. Plastics (1975b), FLAME RETARDANTS 52 (9): 42-4 (September)

Molokhia, M.M. and H. Smith (1967), TRACE ELEMENTS IN THE LUNG. Arch. Environ. Health 15 (6): 745-50 (December)

Molokhia, M.M. and H. Smith (1968), ANTIMONY UPTAKE BY SCHISTOSOMES. Ann. Trop, Med. Parasit. 62 (2): 158-63

Molokhia, M.M. and H. Smith (1969a), TISSUE DISTRIBUTION OF TRIVALENT ANTIMONY IN MICE INFECTED WITH SCHISTOSOMA MANSONI. Bull. WHO 40 (1): 123-28

Molokhia, M.M. and H. Smith (1969b), THE BEHAVIOR OF ANTIMONY IN BLOOD. J. Trop. Med. Hyg. 72 (9): 222-5

Morekas, S. (1975), WASTE VOLUMES AND CHARACTERISTICS FROM THE INORGANIC CHEMICALS INDUSTRY. in: Industrial Waste Management. Seven Conference Papers (SW-156) presented to the National Conference on Management and Disposal of Residues from the Treatment of Industrial Wastewaters, Washington, D.C., U.S. Environmental Protection Agency, pp 47-67 (February)

Murthy, G.K., U. Rhea and J.T. Peeler (1971), LEVELS OF ANTIMONY, CADMIUM. CHROMIUM, COBALT, MANGANESE AND ZINC IN INSTITUTIONAL TOTAL DIETS. Environ. Sci. & Tech. 5 (5): 436-42

Nishioka, H. (1975), MUTAGENIC ACTIVITIES OF METAL COMPOUNDS IN BACTERIA. Mutation Res. 31 (3): 185-9

Norris, J.D. and T.S. West (1974), THE USE OF AN ARGON-HYDROGEN FLAME FOR THE ATOMIC ABSORPTION AND ATOMIC FLUORESCENCE SPECTROMETRY OF ANTIMONY. Analytica Chimica Acta 71 (2): 458-60

O'Brien, W. (1959), THE EFFECTS OF ANTIMONY ON THE HEART. Trans. R. Soc. Trop. Med. Hyg. 53 (6): 482-86

Paint Manufacture (1964), FLAME RETARDANT PAINTS. 34 (4): 47-9

Paton, G.R. and A.C. Allison (1972), CHROMOSOME DAMAGE IN HUMAN CELL CULTURES INDUCED BY METAL SALTS. Mutation Res. 16: 332-36

Portmann, J.E. (1972), POSSIBLE DANGERS OF MARINE POLLUTION AS A RESULT OF MINING OPERATIONS FOR METAL ORES. in: Marine Pollution and Sea Life, Mario Ruivo, Ed., Fishing News (Books) Ltd., Surrey & London, England, pp 343-6

Renick, Abbott and E. Virginia Wright (1951), ANTIMONY. in U.S. Bureau of Mines Minerals Yearbook, pp 151-61

Richtarik, A.A., K. Lindemulder and B. DeBoer (1965), THE ANTIBACTERIAL ACTIVITY OF SELECTED PHARMACOLOGICAL AGENTS. Canadian J. Microb. 11 (4): 637-40

Robitschek, P. and C. Thomas Bean (1954), FLAME-RESISTANT POLYESTERS FROM HEXACHLOROCYCLOPENTADIENE. Ind. and Eng. Chem. 46 (8): 1628-32

Ruch, R.R., V.P. Guinn, R.H. Pinker (1964), DETECTION OF GUNPOWDER RESIDUES BY NEUTRON ACTIVATION ANALYSIS. Nuclear Science and Engineering 20 (4): 381-5 (December)

Sapire, D.W. and N.H. Silverman (1970), MYOCARDIAL INVOLVEMENT IN ANTIMONIAL THERAPY: A CASE REPORT OF ACUTE ANTIMONY POISONING WITH SERIAL ECG CHANGES. South Afric. Med. J. 44: 948-50

Schroeder, H.A. (1965), THE BIOLOGICAL TRACE ELEMENTS OR PERIPATETICS THROUGH THE PERIODIC TABLE. J. Chron. Dis. 18: 217-28

Schroeder, H.A., M. Mitchener, J.J. Balassa, M. Kanisawa and A.P. Nason (1968), ZIRCONIUM, NIOBIUM, ANTIMONY AND FLUORINE IN MICE: EFFECTS ON GROWTH, SURVIVAL AND TISSUE LEVELS. J. Nutrition 95: 95-101

Schroeder, H.A. (1970), A SENSIBLE LOOK AT AIR POLLUTION BY METALS. Arch. Environ. Health 21 (6): 798-806 (December)

Schroeder, H.A., M. Mitchener and A.P. Nason (1970), ZIRCONIUM, NIOBIUM, ANTIMONY AND LEAD IN RATS: LIFE-TERM STUDIES. J. Nutrition 100 (1): 59-68

Semling, Harold V., Jr. (1974), CHEMICAL SHORTAGE -- IT WILL BE WITH US FOR SOME TIME YET. Chem 26 Paper Processing 10 (1): 28-31

Skrinde, Rolf T. (1971), ANALYTICAL METHODS -- ORGANICS. Water Pollution Control Federation Journal 43 (7): 1550-7 (July)

Smith, S.E. (1969), UPTAKE OF ANTIMONY POTASSIUM TARTRATE BY MOUSE LIVER SLICES. Brit. J. Pharmacol. 37 (2): 476-84

Stephenson, Richard M. (1966), *Introduction to the Chemical Process Industries*, Reinhold Publishing Corp., pp 298-300

Taylor, P.J. (1966), ACUTE INTOXICATION FROM ANTIMONY TRICHLORIDE. Brit. J. Ind. Med. 23: 318-21

Taylor, Larry R. and Dennis C. Johnson (1974), DETERMINATION OF ANTIMONY USING FORCED-FLOW LIQUID CHROMATOGRAPHY WITH A COULOMETRIC DETECTOR. Analytical Chemistry 46 (2): 262-6

Thomas, R.G., S.W. Felicetti, R.V. Lucchino and R.O. McClellan (1973), RETENTION PATTERNS OF ANTIMONY IN MICE FOLLOWING INHALATION OF PARTICLES FORMED AT DIFFERENT TEMPERATURES. Proc. Soc. Exp. Biol. Med. 144 (2): 544-50

Thompson, K.C. and D.R. Thomerson (1974), ATOMIC-ABSORPTION STUDIES ON THE DETERMINATION OF ANTIMONY, ARSENIC, BISMUTH, GERMANIUM, LEAD, SELENIUM, TELLURIUM AND TIN BY UTILISING THE GENERATION OF COVALENT HYDRIDES. Analyst 99 (1182): 595-601 (September)

TRW (1973), *Industrial and Municipal Disposal Candidate Waste Stream Constituents Profile Reports* - Inorganic Compounds, Recommended Methods of Reduction, Neutralization, Recover or Disposal of Hazardous Waste, Report No. 21485-6013-RU-00, Vol. XII. pp 175-213, Prepared for U.S. Environmental Protection Agency (1 February)

Wagoner, J.K., R.W. Miller, F.E. Lundin, Jr., J.F. Fraumeni, Jr., and M.E. Haij (1963), UNUSUAL CANCER MORTAILITY AMONG A GROUP OF UNDERGROUND METAL MINERS. New Engl. J. Med. 269 (6): 28-4 (8 August)

Waitz, J.A., R.E. Ober, J.E. Meisenhelder and P.E. Thompson (1965), PHYSIOLOGICAL DISPOSITION OF ANTIMONY AFTER ADMINISTRATION OF ^{124}Sb LABELLED TARTAR EMETIC TO RATS, MICE AND MONKEYS, AND THE EFFECTS OF TRIS (p-AMINOPHENYL) CARBONIUM PAMOATE ON THIS DISTRIBUTION. Bull. WHO 33: 537-46

Wan-chu, Ma, Kuo Kang-wan, and Sung Chen-yu (1959), THE TOXICITY OF ANTIMONYL QUININE HYDROCHLORIDE AND THE ABSORPTION, TISSUE DISTRIBUTION AND EXCRETION OF ANTIMONY AFTER ORAL ADMINISTRATION OF THE DRUG. Acta Pharm. Sinica 7: 144-8

Way, J.J.R. (1975), ANTIMONY. in: *Mining Annual Review*, pp 113-5

Webster, S.H. (1946), VOLATILE HYDRIDES OF TOXICOLOGICAL IMPORTANCE. J. Ind. Hyg. Toxicol. 28 (5): 167-82

Wester, P.O. (1965), CONCENTRATION OF 24 TRACE ELEMENTS IN HUMAN HEART TISSUE DETERMINED BY NEUTRON ACTIVATION ANALYSIS. Scand. J. Clin. Lab. Invest. 17 (4): 357-70

Wester, P.O. (1965a), TRACE ELEMENTS IN THE CONDUCTIVE TISSUE OF BEEF HEART DETERMINED BY NEUTRON ACTIVATION ANALYSIS. Acta Med. Scand. 178 (6): 789-99

Woodruff, A.W. (1969), COMPARATIVE VALUE OF SOME CURRENTLY USED ANTI-SCHISTOSOMAL DRUGS. Ann. N.Y. Acad. Sci. 160: 650-55

Woolrich, P.F. (1973), OCCURRENCE OF TRACE METALS IN THE ENVIRONMENT: AN OVERVIEW. Am. Ind. Hyg. Assoc. J. 34 (5): 217-26

Wyche, C. (1973), ANTIMONY. in: Bureau of Mines Minerals Yearbook, U.S. Dept. of the Interior, Washington, D.C., pp 1-9

Wyche, C. (1975a), ANTIMONY. in: Bureau of Mines Bulletin 667, Mineral Facts and Problems, 1975 edition, U.S. Department of the Interior, Washington, D.C.

Wyche, C. (1975b), ANTIMONY IN THE THIRD QUARTER 1975. Mineral Industry Surveys, Bureau of Mines, U.S. Department of the Interior, 5 pp. (8 December)

Yanagisawa, Masaaki, Tsugio Takeuchi, and Masami Suzuki (1973), FLAMELESS ATOMIC ABSORPTION SPECTROMETRY OF ANTIMONY. Analytica Chimica Acta 64 (3): 381-6

Youson, J.H. (1973), POTASSIUM PYROANTIMONATE AND OSMIUM-ZINC IODIDE REACTIVITY IN THE TUBULAR EPITHELIUM OF THE OPISTHONEPHRIC KIDNEY OF THE SEA LAMPREY. Petromyzon marinus L. J. Morphology 140 (1): 119-33

Zaki, A.A. (1955), A PRELIMINARY STUDY OF THE EFFECT OF INTENSIVE DOSES OF ANTIMONY ON THE HEART. Trans. R. Soc. Trop. Med. Hyg. 49 (4): 385-86